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GLANDS OF THE ESOPHAGUS.*

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The general pathologist has shown very little interest in the glands of the esophagus. Their relative importance is well illustrated by the fact that they occupy only a paragraph, or at most a page, in the voluminous text books on Pathology. Many similar obscure and neglected corners of medicine have proven, on investigation, interesting and of some importance. This has been my experience, in studying this subject during the past ten months.

The purpose of this paper is to record the findings in over one hundred autopsy specimens of the esophagus in Dr. Mosher's Laboratory at the Massachusetts Eye and Ear Infirmary. They include about fifteen hundred sections from the various esophageal levels. The primary object of the study was an attempt to explain the reported biopsy findings of gastric mucosa associated with ulceration of the esophagus. The additional pathological findings are submitted in the hope that they may add something to the comparatively small amount already on record regarding this subject.

The order of the subjects discussed is as follows: a short historical note; the embryological development of the glands;

*All photographs and drawings by the author.

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the histological structure and classification; acute and chronic infections; carcinoma; webs and cysts.

F. A. Schmidt, in 1805, first described the esophageal glands. He did not recognize the two groups but Rudinger and Krause (1897) described the deep glands and another type of tubo-acinous gland superficial to the muscularis mucosa. In 1887, Lautenschlager, after examination of a great number of esophagi was unable to confirm these observations.

The glands were then overlooked until J. Schaffer (Berlin, 1897) again called attention to them. He gives an admirable histological picture of the two distinct types of glands and his article is the best that has been written on the subject. Oppel added the observation that parietal cells may or may not be present in superficial gland areas.

Many more recent articles have been written with regard to the possibilities of these glands as factors in the pathological lesions of the esophagus. Nakamura has spoken of the formation of cysts. Mallory gives the glands as a possible origin of benign adenomata and adeno-carcinomas.

Feldman and Zinn (1929) report a case of gastric mucosa removed from an ulceration at the upper end of the esophagus. They conclude in part: "Gastric heteropia is not rare. Ordinarily the secretion from these is neutralized by saliva. It is possible, however, that acute peptic ulceration with extensive heteropia and accompanied by spasm of the cardia, may lead to the accumulation of considerable quantities of acid secretion and delay healing of the ulcer."

G. Jackson (1929) states that focal infection is the chief cause of peptic ulceration of the esophagus but perpetuation of the ulceration is due to gastric juice. Islands of gastric mucosa at the cardia are believed by him to be a factor in this but that it is an essential factor he believes not yet proven.

Rehfus states that the now clearly proven selective action of bacteria, toxins and even chemicals and drugs, when introduced in various ways, renders of utmost importance the presence of islets of gastric mucosa in the mucosa in the esophagus in the etiology of peptic ulceration.

The embryology can be reviewed also quite briefly. The most rapid development of the esophagus occurs during the fourth week. There is a double origin with the upper or peritracheal division derived from the retropharyngeal segment and the lower from the fore-gut.

In a paper by Johnson, he found the cardiac glands developing

in the 78 mm. (12 weeks) embryo and occurring in their adult position at both the upper and lower ends. These glands are developed at the same time as those of the duodenum. The deep or mucous type are not found until much later and appear in the 240 mm. (28 weeks) embryo. These findings in this work done at the Harvard Medical School preclude the other theories of development of these structures. These were: *1.* That all glands

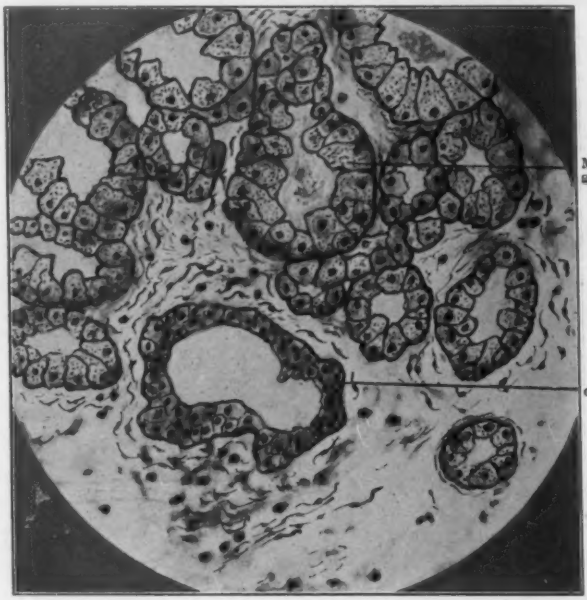

Plate No. 1. Deep or Mucous Glands (Retouched Photo-Micrograph). These are the typical glands of the esophagus. They are mucous in type of tubulo-alveolar structure. The cells stain blue with hematoxylin, have a granular appearance and the lumen is usually filled with mucous.

were originally of the superficial type that changed to mucous with some areas failing to change (heteropic development-Schaffer); *2.* That all are structures developed from the primary basal layer of the entoderm by direct differentiation in the determined areas (Ebert and Schriddle); *3.* That the superficial type develop early and are crowded down by the squamous epithelium, persisting in some areas (Lewis and Stroff). However the development, it is evident that this extra type of gland is embryologically different from the mucous glands, whether at the cardiac orifice or in islands at the upper end.

The histology and classification has been well given by Schaffer. Briefly, there are two distinct types of glands, the deep or mucous and the superficial or cardiac. The mucous or deep glands are the true esophageal variety. They are found throughout the whole length of the structure although more abundant in the upper half. They are always beneath the muscularis mucosa. The gland is tubulo-alveolar in type, having large mucous cells with a peripheral nucleus. They stain blue

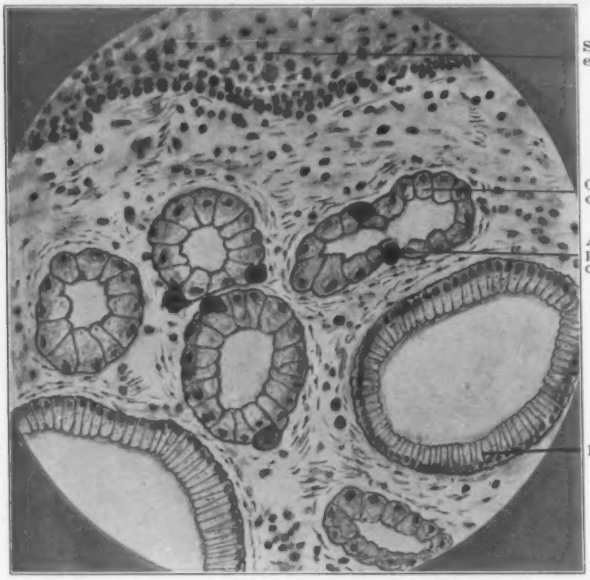

Plate No. 2. Superficial or Cardiac Glands at the Lower End of the Esophagus (Retouched Photo-Micrograph). Glands are branched tubular type with cells containing granules and staining with eosin. They are always superficial to the muscularis mucosa. There may be acid parietal cells attached to the periphery of the alveoli. The ducts are of high columnar epithelium with nucleus at the base, giving a palisade appearance on section.

with hematoxylin and give the typical mucous reactions. The ducts are composed of several layers of pseudostratified cuboidal epithelium (see Plate 1).

The superficial or cardiac glands are the same as the glands of the cardia of the stomach. They are found at the lower end extending for a few mm. above the cardiac orifice (Schaffer). In the upper end they occur as isolated islands eroding the epithelium on the lateral wall as far down as the fifth tracheal

ring. In the lower region they occur either as erosions, in direct contact with the lumen, or immediately beneath the epithelium but always superficial to the muscularis mucosa. They are oval or pyramidal groups of branched tubular type staining with eosin. The ducts are formed of a single layer of high columnar epithelium, the nucleus at the base giving a quite characteristic palisade appearance. Acid parietal cells may or may not be attached to the periphery of the lobule and are not necessary for the diag-

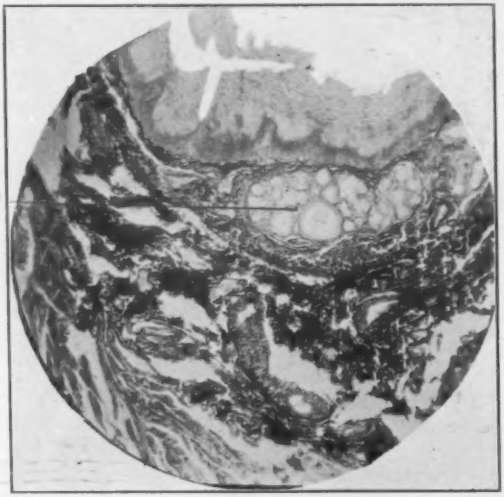

Plate No. 3. Superficial Glands Lower End of the Esophagus. A32-8—Autopsy Diagnosis: Tubular Hemorrhage. Showing glands immediately below the squamous epithelium as they occur immediately above the transition from the cardiac area.

nosis of this type of mucosa in the upper end of the esophagus. The true function of these glands is not known. In spite of their staining with eosin and their appearance, they do give a modified mucous reaction. That they have also some digestive action in their secretion is quite probable (see Plates 2 and 3). In addition to these points we can add that the superficial glands can occur as high as one inch above the cardiac orifice and that cells difficult to distinguish from true serous types can be found (see Plate 3).

With regard to the latter observation we found first, about the middle of the esophagus, a group of deep glands that were very similar to the true serous salivary glands. These had a central

nucleus in a cuboidal type of cell and stained with eosin, and not hematoxylin, as they should. Dr. Tracey Mallory stated that he had never seen glands of this type in the esophagus and that they appeared to be serous. We later found a group of typical mucous type mixed with some apparently serous. Finally there was a section showing, in the same alveolus, side by side, cells of each type. We found this in a number of sections later and con-

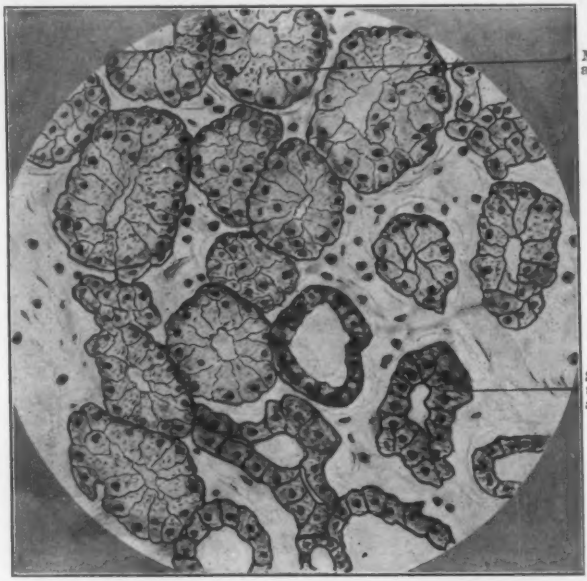

Plate No. 4. Resting Mucous Alveoli—Middle of the Esophagus (Retouched Photo-Micrograph). Certain alveoli that seemed typically serous in nature are seen. These have a central nucleus and eosin-staining cytoplasm in contrast with the blue, mucous-filled alveoli present in the same group.

cluded that this was probably a resting stage, with gland cells empty of mucous. The nucleus became central and the cells were serous in appearance and in staining reaction. No serous glands have been reported in the esophagus but Lewis suggests (1906) that mucous glands may at times, when empty, resemble serous types (see Plates 4 and 5).

The next consideration is infection and its relation to the glands. Frequent reports have been published of ulceration at the upper end of the esophagus which at biopsy have been

reported to show gastric mucosa. This gastric mucosa is in all probability normal superficial or cardiac gland areas that Schaffer was able to demonstrate in the gross in 70 per cent of autopsies. These erosions of the pinkish gland epithelium as seen in contrast with the other mucosa, especially as these are depressed, might well appear as small ulcerations. They can of course be a cause of infective ulceration in that they are a more accessible

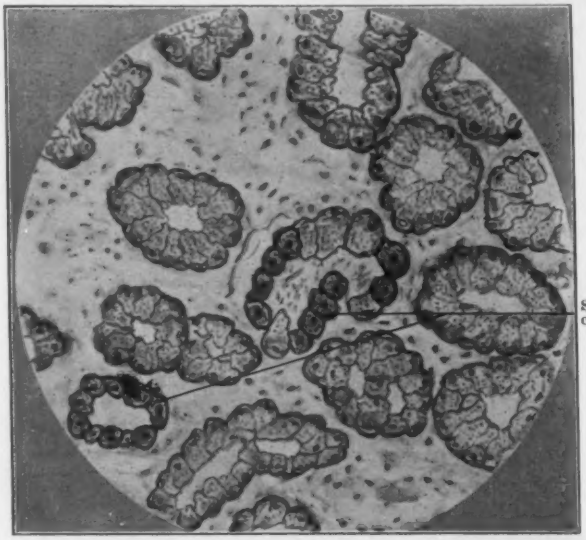

Plate No. 5. Mucous and "Serous" Cells in the Same Alveolus (Retouched Photo-Micrograph). The serous type of cell with its central nucleus is seen in the same alveolus with normal mucous cells. The apparent serous nature is due to the absence of mucous in a resting cell.

point of entry for infection than the protective wall of stratified squamous epithelium. In addition to this, we have the added factor that the secretion may have some digestive function (see Plate 6). We have not had any slides showing infection in these glands in the upper esophagus but have had considerable evidence in those from the cardiac region (see Plate 6).

Infection in the lower end is indeed quite common in the glandular areas. It is more often found in the cases of acute infections that have, or might have, blood stream involvement during their course. We have found all stages from a lymphocy-

tic infiltration around the glands and ducts to abscess and ulceration in the gland areas (see Plates 7, 8 and 9).

Inflammation associated with a moderate cystic dilatation of the glands and the ducts is relatively common. Lymphocytic infiltration alone of gland areas especially around the ducts of the mucous type is similar to that found in mucous glands in any part of the body and is of too frequent occurrence to be of

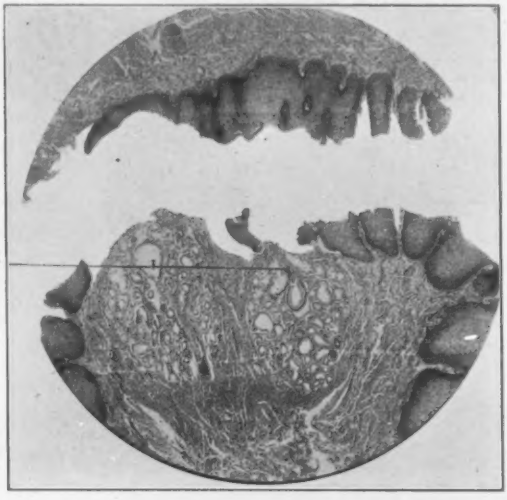

Plate No. 6. Superficial or Cardiac Glands at Upper End of the Esophagus. A31-116 — Autopsy Diagnosis: Neoplasm Left Cerebral Hemisphere. The cardiac glands are here eroding the stratified squamous epithelium as can normally be found in sections from the upper esophagus. These areas are frequently large enough to be recognized in the gross.

any pathological significance. But frequently, with some evidence of cystic changes, one finds infiltration with plasma cells and occasional polymorphs. This must be placed in the group of chronic inflammatory changes. Whether the infiltration was due to the previous cystic change with the retained secretion acting as an irritation, we were unable to determine. This rather than that the cyst formation was the result of a previous infection seems the more likely explanation (see Plates 10 and 11).

Dr. Mosher has previously reported that large cysts may result from obstruction of the ducts by a downgrowth of the stratified squamous epithelium, forming a plug. Since that report we have had several cases showing this type of lesion. Two of

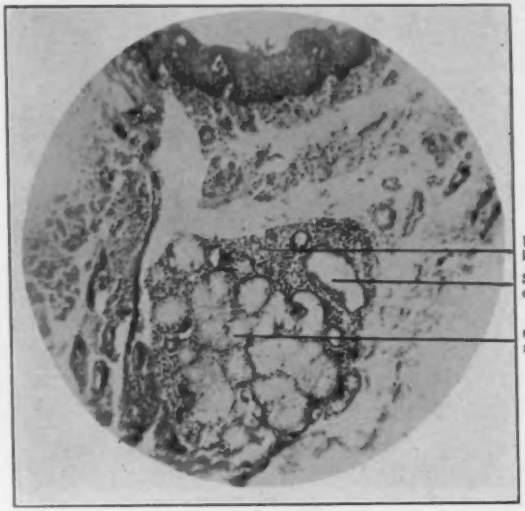

Plate No. 7. Small-Celled Infiltration Around Glandular Group. Frequently an infiltration of lymphocytes is seen, as above, around glandular areas. This seems of too common an occurrence to be termed pathological.

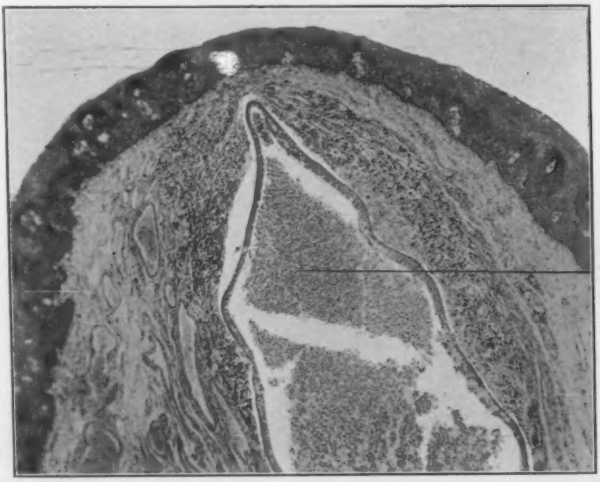

Plate No. 8. Dilated Gland Duct Filled with Pus. A30-116—Autopsy Diagnosis: Cirrhosis of the Liver. Duct has been markedly dilated and is simply a bag of pus.

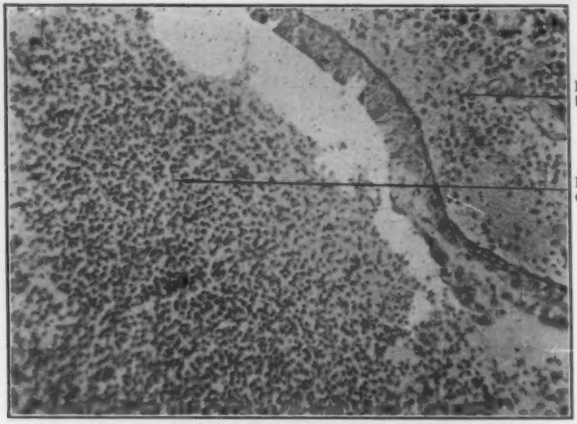

Plate No. 9. H.P. of Dilated Duct with Pus. The purulent exudate is seen not only filling the dilated duct but also a diffuse polymorph. infiltration in the surrounding stroma.

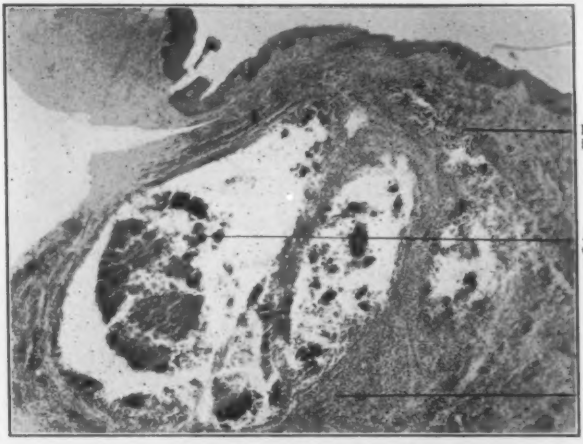

Plate No. 10. Abscess in Esophageal Mucosa. A32-42—Autopsy Diagnosis: Broncho-Pneumonia. There is a central abscess cavity that has the appearance of a dilated gland duct. There is a well formed abscess wall and an intense polymorphonuclear infiltration is seen throughout the neighboring stroma.

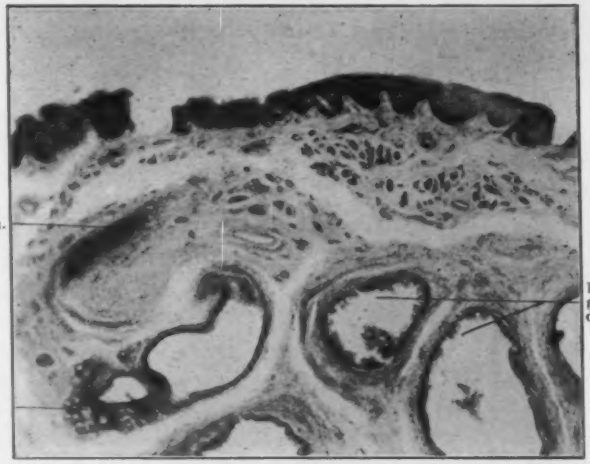

Plate No. 11. Acute Infection in Glandular Areas. Section shows marked dilatation of ducts. There is pus within the cystic areas as well as a diffuse polymorphonuclear infiltration within the gland areas and throughout the adjacent stroma.

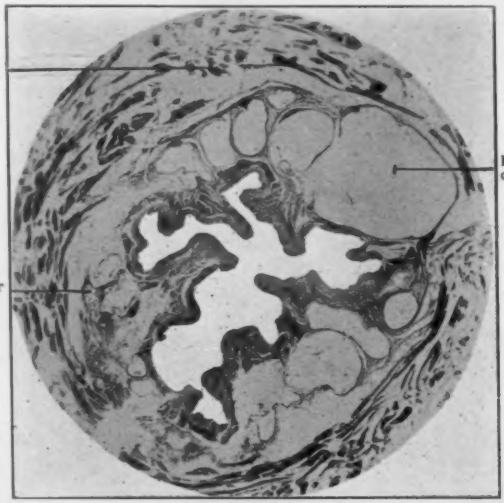

Plate No. 12. Marked Cyst Formation in Mucosa of the Esophagus. A32-34—Autopsy Diagnosis: Cirrhosis of the Liver. There is a marked cyst formation of the gland ducts. These cysts fill the entire mucosa and in places crowd and disintegrate the esophageal musculature.

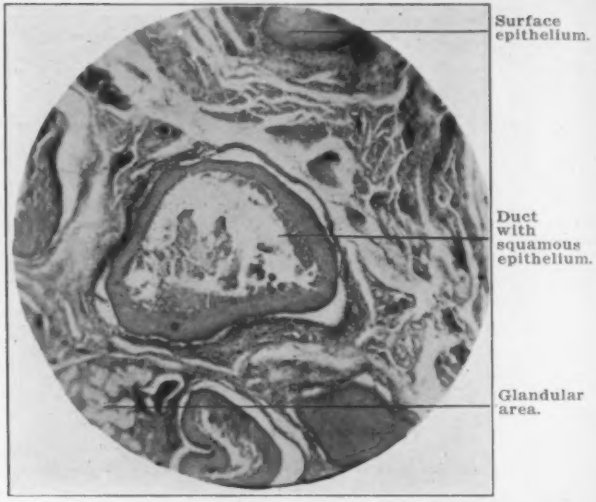

Plate No. 13. Cysts Deep in Mucosa Filled with Squamous Epithelium. A32-34—Autopsy Diagnosis: Cirrhosis of Liver. Section shows the stratified squamous epithelium filling the cystic ducts completely down to the gland acini. Although the cells show marked activity, there is no evidence of invasion outside the duct.

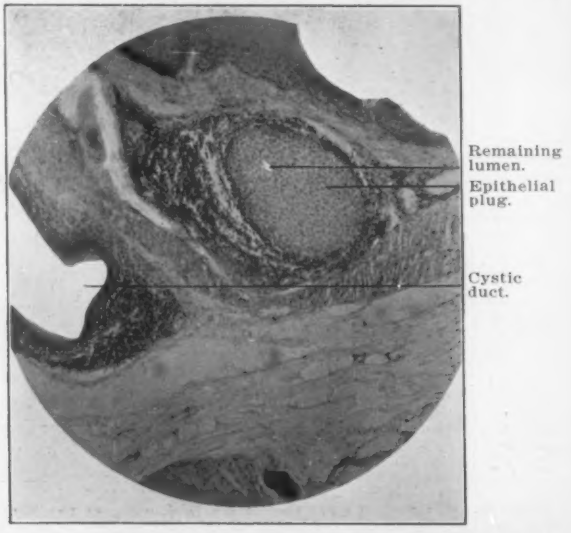

Plate No. 14. Occlusion of Duct by Epithelial Plug. A32-42 — Autopsy Diagnosis: Broncho-Pneumonia. Section shows the small opening left in the dilated duct which is filled by a squamous epithelial downgrowth. There is also seen a dilated portion of the duct below this with marked surrounding infiltration of both areas.

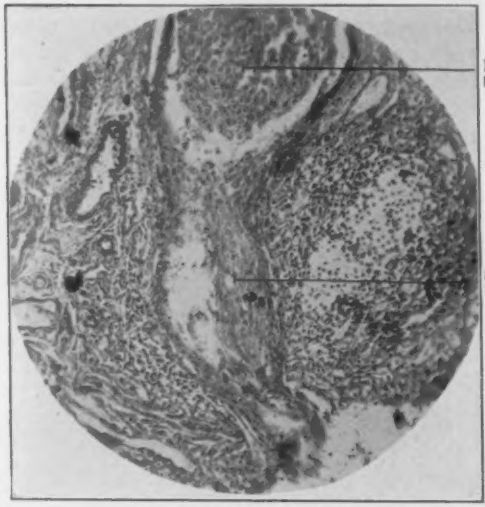

Plate No. 15. Gland Duct Showing Downgrowth of Surface Epithelium. A31-89—Autopsy Diagnosis: Atrophic Cirrhosis of Liver. The duct is cut in longitudinal section and shows the squamous epithelial downgrowth. A surrounding inflammatory reaction is seen.

Plate No. 16. Paget's Disease of the Breast (Photo-Micrograph from Dr. Mallory's Laboratory). Section shows marked dilatation of the gland ducts of the breast with a downgrowth of squamous epithelium. This is usually a premalignant manifestation in the breast.

the outstanding ones have been from cases that had a cirrhosis of the liver. In one of these, the cysts could be plainly seen in the gross, encroaching on the esophageal lumen. On microscopic examination, they are seen to fill the whole mucosa and even to disorganize the circular muscle layer (see Plate 12). The cause of the obstruction is the downgrowth of the stratified squamous epithelium that in some areas almost completely fills the cysts.

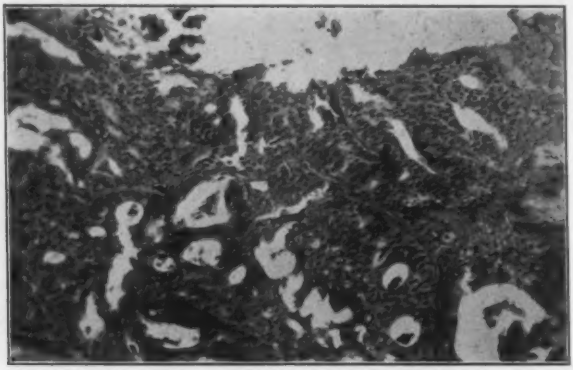

Plate No. 17. Adenocarcinoma. Biopsy No. 968. There is a diffuse glandular hyperplasia with definite malignant change. Section is from lower end of esophagus at the transition of glands.

Although there is no evidence of invasion beyond these areas, the epithelium is of a quite active type (see Plates 13, 14 and 15). Dr. Mallory has noticed a similar condition in the ducts of the breast in Paget's Disease. Here, also, is a cystic dilatation of the ducts and a downgrowth of the surface epithelium. This lesion in the breast has been definitely established as a precancerous lesion (see Plate 16). Whether this is a premalignant manifestation in the lower esophagus, we cannot prove.

Adenocarcinoma does occur in the esophagus. It is, of course, relatively infrequent as compared with the epidermoid type. We have four cases all showing a good attempt at differentiation into glandular types. One can not be certain of the gland type in any of these sections but it is of significance that these were all from the area of transition from the superficial to the deep type of glands. Simple adenomas do occur (Dr. T. B. deep type of glands. Simple adenomas occur (Dr. F. Mallory) but we have no specimens showing this lesion (see Plate 17).

There is one case of a biopsy from a web. This lesion was diagnosed by X-ray and confirmed on direct examination. It

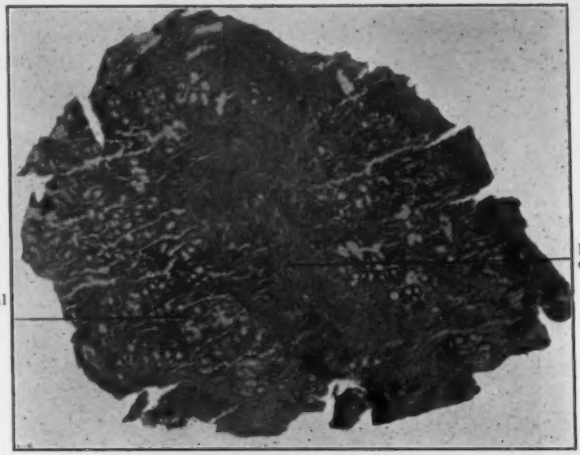

Plate No. 18. Web of Esophagus. Biopsy No. 2298. Section is from a bite taken from an upper esophageal web. It shows a fibrous core but with marked glandular hyperplasia. The glands are typical cardiac glands.

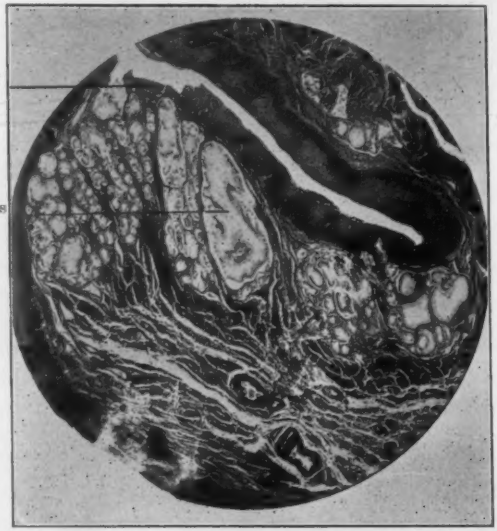

Plate No. 19. Edematous Glandular Areas in Pneumonia. A31-93 — Autopsy Diagnosis: Lobar Pneumonia. This group of superficial glands show marked edema. The appearance simulates the cloudy swelling of liver, kidney, etc., found in toxic disease.

was located at the upper end just below the cricoid. The pathology is interesting in that it showed a fibrous core and many glands with a chronic inflammatory reaction. The significant part was that these glands were entirely of the superficial or cardiac type. That this web was due to infection in this cardiac type of gland, as discussed above under ulceration of the upper esophagus, should be a consideration (see Plate 18).

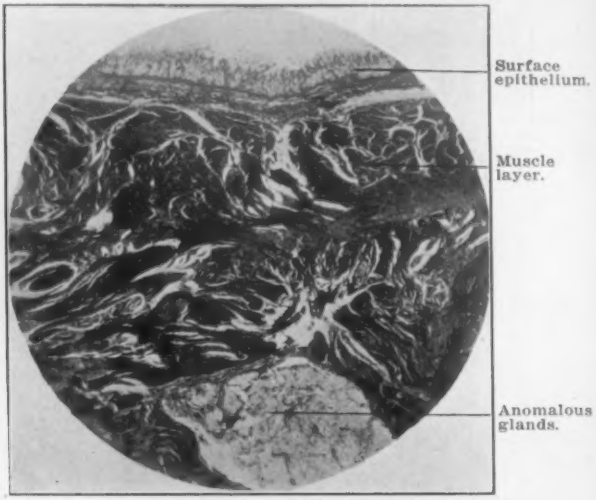

Plate No. 20. Anomalous Position of Esophageal Glands. The group of glands above have burrowed deeply into the esophageal musculature and are found as a nest within the muscle coat. This occasionally occurs in the pharynx.

Various minor changes have been noted in the glands, both superficial and deep. They are definitely affected by degenerative lesions. In myocardial degeneration and chronic wasting diseases they are less in number and stain poorly. One case of pneumonia showed changes that might well be toxic in nature. The glands were of the superficial or cardiac type and appeared quite swollen and edematous. There was no evidence of any inflammatory reaction present. This seems quite analogous to the cloudy swelling of toxic origin found in many acute infections in liver and kidney (see Plate 19).

There was one case of anomalous position of the glands that is of interest only as a rarity. The glands although normal in appearance were found deep in the muscular coat and entirely

surrounded by it. This burrowing into the muscle coat has been reported in the pharynx but not in the esophagus (see Plate 20).

CONCLUSIONS.

1. The primary object of this study was an attempt to explain the reported biopsy findings of gastric mucosa associated with ulceration of the esophagus. I do not believe that true gastric mucosa is ever present in the esophagus. In many cases confusion has arisen from calling the normal superficial glands gastric mucosa. There may occur actual ulceration ingrafted on one of these glandular areas due to the accessibility of their erosions in a lumen that is ordinarily well protected by stratified squamous epithelium.

2. Infection of the glands is relatively common, especially of the superficial type and may range from a chronic to an acute type with ulceration or abscess formation.

3. Cells staining with eosin and similar in appearance to serous glands are found. These are not serous glands but inactive cells emptied of mucous.

4. Cystic dilatation to a minor degree of the ducts of both the superficial and deep glands, is found with no apparent cause. It is frequently associated with a surrounding small-celled infiltration.

5. Large cysts due to obstruction of the ducts are found. These show a downgrowth of squamous epithelium similar to that found in Paget's Disease of the breast which is a precancerous lesion. These cysts are seen in the gross.

6. Four cases of adenocarcinoma were all at the transition of the cardiac and deep glands.

7. Islands of cardiac or superficial glands in the upper end of the esophagus must be considered as a possible origin of webs.

8. Minor changes occur in the glands such as degeneration, in chronic degenerative diseases, and oedema analogous with the cloudy swelling in toxic diseases.

9. Glands may be anomalous in position—burrowing into the muscle layers.

A large number of the slides were checked by Dr. Tracey B. Mallory and Dr. Bradley of the Department of Pathology of the Massachusetts General Hospital. The work was conducted under the supervision of Dr. H. P. Mosher.

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Massachusetts Eye and Ear Infirmary.

IN MEMORIAM

GEORGE FETTEROLF, M.D., Sc.D.

1869-1932.

It is with a feeling of sadness that we announce the death of Dr. George Fetterolf, of Philadelphia, Dec. 29, 1932.

He was a native of Pennsylvania, a distinguished graduate of the University of Pennsylvania of the class of 1887, received his medical degree from this university in 1891, and the honorary degree of Doctor of Sciences from Ursinus College in 1911.

For twenty-three years he was Assistant Professor in the Department of Anatomy, and in 1924 was elected Professor of Otolaryngology at the University of Pennsylvania, a position which he held until the day of his death.

"Dr. Fetterolf contributed many important studies bearing on the Anatomical Relations as applied to Clinical Medicine in its various fields."

He was a member of the editorial staff of the *Annals of Otology, Rhinology and Laryngology* and the *Archives of Otolaryngology*.

He was Vice-President of the American Laryngological Association and would have presided as its honored President at the 1933 session.

Over forty original contributions to the medical literature of his chosen field characterized his special qualifications by the painstaking care, originality and authoritative quality of their presentation.

His scholarly attainments and personal refinement endeared him to his colleagues in the otolaryngological world which he served so efficiently.

M. A. G.

SOME CAUSES AND THERAPY OF OZENA—REPORT OF 135 CASES.*†

DR. CLARENCE W. TREXLER, Honolulu.

Ozena is one of the most difficult chapters in rhinology, in regard to both etiology and therapy. Until thirty years ago, ozena as a definite infectious disease was unknown. Therapy, until very recently, was limited to cleansing the nose. Tertiary lues, chronic sinusitis and rhinoscleroma were all named ozena. Today, there are as many theories of the etiology as attempts of specific therapy for this disease.

Let us now review the most recent theories of the causes of this disease, giving the bacteriological findings and results of treatment of 135 cases in the Allgemeine Policlinic, Vienna, many of whom were under my observation and whose clinical histories were at all times available.

The separation of ozena from other nose diseases, especially from paranasal sinus disease, has evoked much discussion. Even today, Hajek[1] says it is a false conclusion when many authors make a differentiation between ozena with accessory sinus infection and ozena without accessory sinus infection.

Recently, G. Berberich[2] viewed ozena as neither clinically nor bacteriologically a disease entity, regarding anatomical, constitutional, endocrine and trophoneurotic factors as being essential, if not practically of decisive importance.

A. Glasscheib[3] sees the etiology as being due to the lack of A and D vitamins in the fetus or early childhood, leading to atrophy of the mucous layers of the nose and throat. By animal experimentation he proved that the lack of these vitamins not only diminished resistance to infection directly, but also lead to increased alkali content of the nasal secretion, thereby giving bacteria good occasion for development in the nose.

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*Read before the Hawaii Territorial Medical Association at its annual meeting in April, 1932.

†This treatise is made possible through the co-operation of Prof. H. Marschik and Prof. Schnierer, of the Allgemeine Poliklinik.

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Halphane and Schullmann[4] reduce ozena to a sympatheticoendocrine disturbance in the suprarenal, thyroid, parathyroid and sexual glands. They have reported success in therapy by adrenalin, hypophysis and ovarian extracts.

Schnierer,[5] upon the incidental disappearance of the ozena odor during the treatment of pernicious anemia by liver, resolved to make further surveys. In fifteen cases of severe ozena he gave liver preparations—hepatopson and procytol. He used ventraemon, a stomach wall preparation in ten cases. Odor disappeared in each case, but upon the interruption of therapy quickly returned.

Some investigators believed that the excessive roominess in the nose itself was the essential factor in causing crust formation, as the secretion could not be expelled. To that end, injections of paraffin and allied substances in submucosa of the septum and inferior turbinates were tried. This is less in favor today. For the same condition autotransplants were made in the nose in attempting to make the airways conform to normal proportions. Ozena was still to be reckoned with.

The belief that bacteria play the chief role in the etiology of ozena has been steadily increasing. This has led to numerous attempts to use specific therapy and it has, therefore, prompted the investigations and determined the therapy of the case in this report.

Investigation of ozena has had an important stage in the finding by F. Perez[6] of the coccobacillus fetidus in the bacterial flora of the nose in typical ozena cases. This led to the view of the bacterial etiology, receiving added impetus by the experimental findings of G. Hofer[7] and E. Frankel.[8] Abel[9] and Lowenberg[10] found the bacterial preponderance of B. mucosus (Abel) and noted the similarity of that organism with B. pneumoniae of Friedlander.

As first investigator, credit is given to Perez for finding a bacillus which only occurs in the nose of genuine ozena patients, but does not occur as a saprophyte in the bacterial flora of the nose. He was also the first to prove specificity of the coccobacillus fetidus for ozena through his animal experimentation. By injection of pure cultures of those in rabbits, he was able to produce a sanguineous effusion of the bones of the turbinates, suppuration of the mucous layers and disappearance of the bony parts of the turbinates. Those pathological changes are analogous to findings in ozena. From the nose of each of the inoculated animals he was able to recover the organisms and produce a culture. Likewise, G. Hofer was successful in producing a nasal ozena-like infection by bringing the infective material into the nose of man.

The coccobacillus fetidus was described by Perez. It grows blue on Drigalsky's nueritive media, and produces, in bouillon cultures, the typical sweet, fetid odor of the ozena nose. He found that this organism occurred as a saprophyte in noses of dogs having distemper disease, and he concluded that infection from dog to man was possible as well as from man to man.

In one series reported by Perez, he found the coccobacillus fetidus in seven out of eleven cases, or in 63.6 per cent. In this series that organism occurred in but about 9 per cent. Intravenous injection of cultures in dogs failed to give satisfactory results in recovery of the organisms from the nose of the animals as claimed by certain investigators. In all cases of typical ozena, crusts were removed from the noses and bacteriological investigations were made by the Vienna State's Serological Control Station, under the supervision of Prof. Busson. He reported the finding of the following bacteria: B. mucosus ozena (Abel), B. Proteus vulgarius, B. pseudodiphtheria, bacilli of coli group, B. fluorescens, hay bacillus, different Gram negative bacilli, streptococci, staphylococci, pyocyaneus, different cocci suspicious for coccobacillus fetidus and finally, coccobacillus fetidus itself. The relatively small percentage of finding the latter organism may be explained by the fact that cases came in chiefly with advanced stages of ozena in which that sensitive organism may have been overcome by other bacterial flora. Hofer made a vaccine out of this organism but only a relatively small number of cures were effected. It is apparently true that the invading organisms remain but a short time. Marschik and Schnierer[11] take the view that most of the cases of developed ozena are either progressive or end stages of an infectious disease, in which the primary bacteria have been replaced by secondarily invading bacteria of various types. It is noteworthy that in 63 cases of the 135 of this report, the B. mucosus (Abel) was present (33 in pure culture, 30 with other bacteria). Marschik[12] believes that these bacilli could localize in ground prepared by the coccobacillus fetidus, thereby playing an important role in maintaining the ozena process.

It was noted that there was another organism, B. pneumoniae, Friedlander, which resembled B. rhinoscleroma and B. mucosus in the biological, cultural and morphological aspects. These three organisms were included in the Marschik-Busson vaccine, called mixed capsule bacteria vaccine, as each has a capsule. Marschik was the first to describe and use this triple vaccine. As early as 1912, Cobb and Nagle[13] had success in treatment of 90 cases of ozena with B. mucosus (Abel) monovaccine. D. Styl Demetriades[14] likewise re-

ported success with that vaccine. Rebattu and Proby[15] as well as Viggo Schmidt[16] used that vaccine, but in too few cases to have any considerable results. Rebattu and Proby used the vaccine in the form of a spray upon the nasal mucosa, subsequent to scarification, and claimed to have made some progress.

The vaccine largely used in this report was the mixed capsule bacteria type introduced by Marschik as mentioned above. Unlike the coccobacillus vaccine, it is impossible to make this vaccine containing a given number of bacteria. Through a special method, the capsules are destroyed to make free the albumin content. Then they agglutinate and cannot be counted. Therefore, this vaccine is made in six concentrations, the first being a concentrated emulsion. Dilutions are made 1:10, 1:20, 1:40, 1:80 and 1:160. In therapy, start by giving semiweekly injections of the 1:160 dilution for four doses. Then administer four doses of the next higher concentrated emulsion. Repeat until you have reached the concentrated emulsion. In each case, 1 cc. is the amount given in the arm intramuscularly. After twenty injections, you reach the concentrated emulsion, and, if the patient has borne the therapy well, you may give gradually increased doses but in no case stronger than 0.5 cc. of the concentrated emulsion with the same amount of physiological salt solution at a single administration.

REACTIONS.

After injection, three kinds of reactions may occur: focal, local and general. Focal reaction consists of crust desquamation in the nose, increased secretion, sometimes bleeding of the mucous layer and appearance or disappearance, increase or decrease of the odor. These may occur after the first injection and are usually more marked during the first twenty injections. Especially the change of odor is of great interest, for in many cases in which the odor has been absent for a long time prior to treatment, returned within the first five injections, or the odor, permanent before beginning therapy, disappeared after the second or third injection and then appeared intermittently until finally there was no recurrence.

A local reaction usually does not make its appearance during the injection of the first four concentrations. If it is then present, it consists of but slight pain in the injected arm. Marked allergic symptoms may start with injection of vaccine of the 1:10 concentration and increase with the stronger doses. In spite of it, interruption of therapy was not indicated. Marked allergic signs were large areas of infiltration, sometimes over the whole arm, redness, edema, but never suppuration.

The general reaction sometimes begins with the first injection, consisting of headache, slight increase of temperature accompanying it. The fever may reach 103° F. after injection of the concentrated suspension. Only in a single case was a certain kind of anaphylactic shock seen and this did not recur subsequent to further injections. While in general, therapy is well supported, it is impossible to forecast the numbers of injections which will suffice in a given case. This doubtless is influenced by the stage of advancement of the ozena or by individual factors.

RESULTS OF THERAPY.

This report covers 135 treated cases who were representatives of nine countries—a cross-section of the continent. There were 28 males and 107 females, the ratio of 1:4, which is thought to be about the usual finding. The ages ranged from 6 to 78 years, the average being 26.9 years. The vast majority came in after the disease had become progressively worse for several months, or "Balkanized" we might say. Many of the cases are still undergoing treatment. Hence this report is not the final result. The Marschik-Busson mired capsule bacteria vaccine served as the basis of therapy. The number of injections varied from 10 to 102. After completion of treatment, 10 had noses of absolutely normal aspect and can be accepted as healed. Of that number, four have been observed for an average of four years subsequent to treatment, and they remain normal. The other six have been observed a shorter period, during which time two recurrences have been observed, but they were quickly improved by short, new injection series. So it appears that 10 cases, or about 7.5 per cent of those treated, may be considered normal at this time. Of the other 125 cases treated, 99, or approximately 80 per cent, showed marked improvement with decreased crust formations and odor, but the lowest degree of improvement was the atrophy. But here must be recalled the fact that a very large per cent had marked atrophy of the turbinates upon admission to therapy. At the conclusion of treatment, 92 cases are recorded as having lost the odor entirely; 11 cases have been treated too short a time to warrant any conclusions as to the benefit of treatment; 14 cases, or 11.4 per cent of the longer treated patients, apparently showed no improvement by this therapy.

It is well to note that several of the much improved cases, as well as a number of the healed ones, did not show any change for the better until the fiftieth injection. The continuation of treatment finally brought a rather sudden improvement until even complete

healing took place. In three other cases in which the coccobacillus fetidus (Perez) could not be cultured from the crusts of ozena noses at the beginning of treatment, it was found that during therapy, by Marchik-Busson mixed capsule bacteria vaccine, the above organism appeared. All three cases were well influenced by the therapy.

OTHER FACTORS CONSIDERED.

There were 34 cases, or 25.2 per cent, who also had tuberculosis. But it generally is believed that tuberculosis plays no other role in the pathogenesis of ozena than in decrease in the general resistance against other infectious diseases. Of greater significance is the fact that in 35 cases, other members of the family were suffering from ozena. It is likewise interesting to note that 35 of the cases had dogs as pets in their homes. Whether the contact with such animals really plays a part in development of ozena in man may be an open question. It is noteworthy that four of the cases having advanced ozena were employed in a tobacco factory. These reported that many other workers in the same plant suffered from a diseased condition of the nose not unlike their own. Finally, I give great importance to the finding of a large face with rather short sagittal diameter of the nose, and developed saddle nose. This typical chamaeprosopy was present in 54 cases, or 40 per cent, while 32, or 24 per cent. had saddle noses. It was found that leptoprosopy (small face with rather long sagittal diameter of the nose) was present in but 15 cases, or 11.1 per cent. The other cases, about 49 per cent, assumed an intermediate form. In consequence of the preponderance of chamaeprosopic skull formation in this series, and likewise the fact that just these cases seemed to show the severest ozena process, and the fact that in these cases a large number of pure cultures of bacillus mucosus (Abel) were found, it would seem just that the constitutional importance of skull formation, wide nose and short diameter of the nose in the anterior, posterior direction, should have received wider recognition. However, Hofer, who has done much research in this field, had this to say: "None of the congenital, predisposing factors have more than value of speculative deduction and not the slightest proof * * * we should not give them an etiological importance." Nager's[17] theory of congenital disposition and Siebenmann's[18] theory of the importance of the wide nose merit close study. For the last speaks, almost without exception, of the unequal development bilaterally of the ozena process in the presence of septum deviation, the greater pathological area usually being in the nostril having the greater room.

OTHER ATTEMPTS AT THERAPY.

Mixed capsule bacteria vaccine locally in the nose and a mixed antivirus was also locally used, local irritation was secured by painting the nasal mucous layer with a phenol preparation, "Isophenol," all with little success. Schnierer also tried, as unspecific therapy of ozena, the most effectful fever therapy, such as Wagner-Jaureg introduced in the treatment of paralysis. It was the malaria therapy and this was used in four cases, three of whom had a negative Wassermann test and one positive. These patients were infected with tertian malaria organisms, 4 cc. of blood having been obtained from an infected multiple sclerosis patient during an attack. Injections were massaged into the scarified skin. Until this form of treatment was instituted, all four cases had been treated with mixed capsule bacteria vaccine without success. It was found that after the first attack the odor disappeared, but in three cases returned immediately after the last (tenth) attack had subsided. Three cases were now easily to be influenced by capsule bacteria vaccine, losing odor and crusts. Pharyngitis sicca and the atrophic areas showed improvement.

SUMMARY.

Various theories of the etiology of ozena have been advanced, resulting in numerous attempts at therapy. All merit our careful consideration.

From the standpoint that ozena is a chronic, infectious disease which the patient often acquires under a hereditary, constitutional predisposition, attempts have been made with a view of controlling it by specific therapy.

In reviewing a series of 135 cases of ozena treated by injections of the Marschik-Busson mixed capsule bacteria vaccine, we have found the results encouraging, comparing very favorably with other methods of therapy at our command.

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NASAL OBSTRUCTION IN CHILDREN DUE TO SEPTAL ABNORMALITIES: WHAT SHALL WE DO FOR THEM?

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In the light of our present knowledge of the development of the nose, no more perplexing or important problem is presented to the rhinologist than that of determining what to do, when to do it or what advice to give when a child, suffering with anterior nasal obstruction and rapidly developing deformity, is brought to him for relief.

The situation is altogether different from that presented by the adult whose nose is obstructed or deformed owing to distortion or other abnormality of the septum. Here we know that the organ has reached the limitations of growth and development and that the problem, in his case, is chiefly a mechanical one to be solved by a suitable operation; we are not handicapped by enforced precautions, so necessary to safeguard developmental factors when dealing with nasal obstruction in children.

I have, for many years, demonstrated to students and before professional societies the fact that the symmetry of the nose and its proper function, as a respiratory channel, are largely dependent upon normal development of the septum.

Deviation of the septum or destruction of its cartilaginous or bony constituents in early life by traumatism, infection or disease, is a matter of very serious import.

It should be thoroughly understood by those who first see the child after an accident in which the nose is injured, when abscess of the septum is threatened or when a destructive disease is discovered, that there are at that time no physical indications of the dire results to both the shape of the nose and its function that are certain to follow within a few weeks or months and that usually increase in severity up to the age of puberty. These observations are drawn

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from my own personal clinical experiences extending over a period of many years. My deductions are based upon a careful analysis and evaluation of the clinical facts presented by a large number of patients seen and treated during this period.

I know that from earliest childhood the nasal septum is normally under considerable vertical pressure and that this is necessary in order to elevate the flattened nose of the infant into the more prominent organ of the adult. Conversely if, at this early age, the septum is thrown out of the vertical by traumatism, or if its structural material (cartilage and bone) is destroyed by infection or disease, the nose will be undeveloped, twisted or mis-shapen in some manner and there will be nasal obstruction.

Fig. 1. Patient 12 years of age, showing deformity due to fracture of the nose when she was two years old. Septum was crushed and subsequently exerted no developmental force.
Fig. 2. Profile of same patient (before operation).

The slight injuries of childhood contribute largely to the deflected septa of adult life. When the septum is thrown out of the vertical while it is under pressure, it is practically impossible for it to recover its upright position. The deflection may at first be imperceptible, but it increases and soon becomes clinically demonstrable. At the same time the external nose becomes twisted, deflected or depressed, according to the character of the septal defect and the direction in which its developmental force is diverted.

Septal deviations occur in 75 per cent of Caucasian adults, but are seldom found in negroes or Mongolians, whose broad, flat noses constitute a prominent characteristic of these races. This indicates clearly that here the septum has not been called upon to lift the

nasal bridge; the septum therefore being under little or no vertical tension during the developmental period, was not deflected by the usual traumatic injuries occurring during childhood.

If, therefore, the septum is of such vital importance in the development of a normal nose, it is certain that if it is destroyed or its developmental force is misdirected, as shown by deflections, during the years of active growth, the nose will not only be deformed, but, because of loss of function as a respiratory passage, the health of the child will be impaired. It is essential, therefore, that early relief be afforded. It is equally as important that in operating one should bear in mind the fact that the tissues involved are essential to further development of the organ and that, so far as possible, they must not only be preserved, but they must be so placed that their developmental force shall be directed in the proper direction.

Fig. 3. Same patient (age 15) three years after operation.
Fig. 4. Same patient (age 15) three years after operation.

ABSCESS OF THE SEPTUM.

Abscess of the septum is one of the most frequent causes of saddle-back deformity, for it occurs more frequently in childhood than in adult life. Its destructive influence upon the cartilage and bone of the septum is pronounced and thus the nose is deprived at an early age of its most important developmental factor.

Abscess is usually due to traumatism, in which case it is always preceded by hematoma. If this is recognized at once by the attending surgeon and free drainage instituted, followed by strict asepsis, the development of the abscess will be forestalled in the large majority of cases and subsequent deformity will be averted.

The importance of early recognition of septal hematoma cannot be overestimated.

REPORT OF TYPICAL CASE OF ABSCESS OF SEPTUM.

The following case, now under treatment, has been duplicated by many that I have seen; it demonstrates the practical value of the above remarks:

A well-developed boy, eight years of age, the son of a prominent dentist, was brought to me on account of almost complete nasal obstruction and a developing saddle-back deformity.

Two years ago he fell, injuring his nose. For several days he had a profuse nasal discharge. The family pediatrician thought that he had only a bad cold and treated it accordingly.

A week later the boy had a chill and became quite ill. A rhinologist, who was now consulted for the first time, immediately made a diagnosis of septal abscess of an unusually virulent type.

The child was sent at once to a hospital and the abscess opened and drained. After a convalescence, which was greatly prolonged, owing to the severity of the infection, the child recovered, but both nasal passages were almost completely obstructed by a thick, bulky septum.

Six months later the parents noted that the nasal bridge was broader and flatter than it should be. This deformity and the nasal obstruction had gradually increased up to the time that I saw him, which was about two years after the original injury to the nose.

Examination showed a flattened nasal bridge and the nose almost completely obstructed by a very thick septum.

In operating upon this patient I removed submucously a mass of scar tissue from between two very thin laminae of cartilage. I then brought the latter close together in the median line, thus reconstructing the septum as nearly as possible and preserving its structural elements. At the same time this procedure opened up the nasal passages.

I have every reason to believe that the septum will now perform its normal function in the development of the nasal bridge.

In connection with this case it may be well to note that submucous resection of the septum in a patient of this age would have left the nasal bridge without support and would have destroyed all

hope for normal nasal development through the influence of the septum. In such a case as this no constructive tissues remaining in the nose should be sacrificed. If the septum, reconstructed as I have described, fails to develop the nose properly, the defect may be corrected later on by means of conjoined bone and cartilage transplantation, an operation devised by me many years ago and which I have used with great satisfaction in correcting depressed nasal deformities.

FRACTURES OF THE NOSE DURING CHILDHOOD.

The nasal bones and the nasal processes of the superior maxillae are fractured far more frequently than is generally supposed. Often these fractures are not recognized because the usual methods of making a diagnosis are not available on account of the small size of the bones involved and the difficulties incident to examination of injuries in this locality, especially if the tissues are badly bruised and swollen.

In older children the X-ray is of some assistance, but in the very young it has no practical value in making a diagnosis.

My bridge-splint, which I have found invaluable in the treatment of fractures in adults, cannot, for obvious reasons, be used in the case of children.

A severe traumatic injury to a child's nose should always be regarded as a fracture and should be treated accordingly; that is, by manipulating the parts so as to get them into a normal position and then fixing them in that position by means of proper splints. If we can succeed in keeping the parts immobilized for four or five days, we may be reasonably certain that fixation has occurred; for healing, as we all know, occurs with remarkable rapidity in this locality.

In treating these cases I have found my gold-wire splints very useful.

Where there is a well-developed septal deviation, or where there is nasal obstruction and deformity, due to an old fracture that was not properly set at the time of the injury, operative measures for their relief are always indicated.

Several years ago I devised a modified submucous operation for the correction of septal deviations in children. This operation has been published elsewhere and will not be described here in detail; its object is to relieve the obstruction to nasal respiration and to restore the septum to its median position without the sacrifice of any

appreciable amount of its structural material. In this operation the mucoperichondrium is elevated on only one side and the only tissue removed is a very narrow strip of cartilage along the apex of the deflection.

This method of treating these perplexing and difficult cases has been in use for a sufficient length of time for me to note in some of my old cases that there has been no interference with the development of the nose.

SUMMARY.

The following conclusions have been drawn from my personal clinical experience and I feel that their general acceptance will do much towards limiting the number of calamitous results that follow the neglect of the injuries to the nose that occur so frequently during childhood:

1. The septum is a structure of paramount importance in the development of the nose and is under constant vertical pressure during the years of active development. If, owing to even a slight injury, it becomes deflected at this time, it cannot recover its former upright position; the deflection increases and nasal obstruction and deformity are the results.

2. Abscess of the septum is one of the most frequent causes of nasal obstruction and deformity.

This affection is of traumatic origin and is preceded by an hematoma. If proper treatment is instituted at once, the dire results that would follow the impending abscess may frequently be avoided.

3. All corrective operative work upon the child's nose should have for its prime object the preservation of the tissues composing the structural framework, otherwise there will result obstruction to nasal respiration and the nose will appear unfinished or deformed.

2 East 54th Street.

SOME CLINICAL OBSERVATIONS IN LABYRINTHITIS.*

DR. DANIEL S. CUNNING, New York.

Although the first definitely accepted studies of the human labyrinth began to filter through our medical literature many years ago, that organ still continues to surround itself with a peculiar, almost majestic mystery which may, in some respects, be likened to the famous riddle of the Sphinx. The final and complete understanding of both of these mysteries still remains in doubt and, in both cases, at least some diametrically opposite views are steadfastly adhered to by their respective champions. It is an accepted fact that a multiplicity of opposing theories, on any subject, indicates indecision and a probable cause for further study. Personally, I feel that the human labyrinth still remains, to a degree, in this category. Therefore, if in any statement I seem to disagree with any previously accepted theory, I trust I may be forgiven, for the obvious reason that my ideas are purely personal, not necessarily substantiated by other and more experienced investigators.

Two great physiological functions seem to rest, to a large degree, in the labyrinth; namely, equilibrium and hearing; and it is this latter auditory function which has resulted in that brilliant array of publications by our confreres in otology. Such names as Neuman, Mosher, Friesner, Cahill and many others will always stand out whenever and wherever the labyrinth may be discussed.

Almost from the first mention of the labyrinth, it was recognized that this organ was susceptible to pathological changes of great importance and, since it is a very complicated structure, it naturally follows that these pathological changes may vary as to type, cause or degree. There are three common conditions in which the normal function of the labyrinth is definitely disturbed:

1. In traumatism, as in a fracture of the skull with injury to the cochlea, vestibule or semicircular canals. The symptoms are

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naturally extremely varied and usually very violent in their manifestations.

2. When by over-stimulation of the nerve endings, by the endolymph, the individual loses, by greater or less degree, his sense of equilibrium, and frequently shows the associated nausea, vomiting and vertigo, best exemplified by sea or car sickness.

3. A toxic involvement of the labyrinth, which is the result of infection in or around the labyrinth.

The traumatic and irritated labyrinthides are too obvious and self-explanatory to be of particular interest to the otologist, and I shall therefore limit myself to a discussion of labyrinthitis as a result of infection. That is toxic or suppurative labyrinthitis.

Toxic or suppurative labyrinthitis has been variously classified and divided into many different groups, such as Circumscribed, Diffuse, Serous, Suppurative, Manifest, Latent, Meningeal, Tympanic, Metastatic, etc. It seems rather unfortunate that these classifications are so complicated as to be almost overwhelming to the average otologist. It is not my intention, however, to question the brilliancy or correctness of these classifications, but rather to attempt a classification more easily understood. A division based upon the location of the infection seems to offer a simple solution. This division would work out as follows:

1. Perilabyrinthitis: A pathological condition showing labyrinthian symptoms in which the actual infection is not in the labyrinth proper, but rather in the bony wall surrounding it.

2. Localized labyrinthitis: A pathological condition in which the involvement is limited to a portion of the labyrinth.

3. A diffuse labyrinthitis: In which the entire labyrinth is definitely affected.

To consider the subject according to the above classification we must first discuss perilabyrinthitis, the least dangerous, which is ushered in by unexpected and repeated attacks of vertigo, with periods of apparent normalcy intervening. The attacks are usually of short duration, lasting in most cases from a few minutes to an hour. These attacks are nearly always repeated many times, and may be brought on by any sudden head movement, or may occur without any apparent outside stimulus. During the attack the patient shows a rotary nystagmus, usually directed to the diseased ear. The fistula test is absent. The acute symptomatology naturally

prevents a testing of the static labyrinth, but if this is done, an increased rotation and caloric reaction can be obtained. The hearing during the attack is impaired. Probably the most important fact in reaching a diagnosis of perilabyrinthitis is the history showing the frequency of these attacks, and the very important remaining factor that in the interval between them the labyrinth is apparently normal, both as to functional tests and hearing. The condition is associated with an acute mastoiditis, or an acute exacerbation of a chronic ear. In general, the type of mastoid involvement should determine the treatment, which should be limited in all cases to a simple mastoidectomy, or, in the chronic ears, to a radical mastoidectomy. Surgical interference with the labyrinth proper is absolutely contra-indicated.

Second, localized labyrinthitis is an actual suppuration of a portion of the labyrinth. To qualify as a local labyrinthitis, it is necessary to prove that one of the two main divisions of the labyrinth has no serious involvement. We find, in this condition, that while the static labyrinth is seriously impaired, the cochlea remains without any marked involvement, or vice versa. In reality, the common type seen shows a suppurative process to be limited to the static labyrinth, while the cochlea retains a fair percentage of its functions, as elicited by testing the hearing. The invasion usually begins with a necrosis of one of the semicircular canals, most frequently the external horizontal. This invasion frequently produces a fistula and a resulting sub-acute inflammation in the adjacent parts of the canals, a picture not uncommonly seen in the large ear clinics. The condition is practically always associated with a chronic running ear, and the acute symptoms show a spontaneous rotary nystagmus toward the sound ear with a marked vertigo and ataxia. The attack is of fairly long duration, usually lasting for at least one week, and frequently much longer. Since the cochlea is not to any degree involved, the patient usually retains a fair amount of hearing, and this is the one important factor in differentiating it from a diffuse labyrinthitis. After the acute attack has subsided, it is followed by the latent or quiescent period, in which the patient is no longer conscious of any static or vertigenous symptoms, and the hearing is usually fairly well retained. On functional examination, since there must be a partial destruction of the static labyrinth, it is usual to find a corresponding reduction in its irritability, as elicited in the rotation test, by a greatly shortened after-nystagmus in the direction of the diseased ear, and the caloric test showing reactions of shortened duration and reduced force. An important point to be borne in mind is that, so long as the cochlea remains fairly normal,

as shown by a fair retention of hearing, the condition is not nearly so prone to produce meningitis. A localization of the infection to the cochlea is extremely rare, but if present, will show a total loss of hearing with a comparative normal static labyrinth. The picture resembles a severe nerve deafness, and probably cannot be differentiated from it. Since a localized labyrinthitis is practically always associated with a chronic mastoiditis, a radical mastoid operation should be performed after the symptoms have quieted down. Under no circumstances should the labyrinth be opened, or in anywise interfered with, even in the face of a fistula.

Third, when or if, a localized labyrinthitis spreads to invade the entire labyrinth, a generalized condition is naturally produced which is called a diffuse suppurative labyrinthitis. This condition is most commonly seen associated with a chronic mastoiditis, particularly in the presence of a cholesteatoma. It may, however, occur during an acute mastoiditis or may be traumatically produced by ear surgery, a happening which, unfortunately, is not uncommon. The usual portal of entry is through a fistula in one of the semicircular canals, although it may enter by way of a small blood vessel of the labyrinth itself. Other portals of entry are the oval window, as seen when the stapes is dislocated, which may occur during a myringotomy or a radical mastoidectomy. It may also enter through the round window or may result from a labyrinthian invasion from a deep-seated extradural abscess on the posterior surface of the petrous pyramid. Practically all authorities agree that a cholesteatoma is the most frequent cause of labyrinthitis, Jansen stating that in a series of 120 cases of suppurative labyrinthitis, cholesteatoma was the apparent cause of 71. As above stated, the condition is more frequent in a chronic ear infection, but when found during an acute mastoid, a meningitis is much more likely to result. I believe this is due to the fact that in an acute mastoid there has not been sufficient time for a protective walling off process to have occurred.

In this very important condition there are two different stages: the early, or irritative stage; the latter, or quiescent stage. In both stages, however, the entire labyrinth is involved. In the first, or irritative stage, the patient shows the cochlea involvement by a very marked or total loss of hearing. The static labyrinth shows a sudden, violent irritative condition with marked rotary vertigo, loss of equilibrium, spontaneous rotary nystagmus usually directed toward the sound ear, marked dizziness and often nausea and vomiting. The fistula test can frequently be obtained. The total loss of hearing plus the violent static irritability makes the diagnosis

reasonably certain. No functional test in the nature of rotation or caloric should be attempted during the active stage, since to do so is not without actual danger. The vertigo is of such type that when described by the patient, surrounding objects seem to rotate in the same plane as the nystagmus. The patient practically always seeks the comfort of bed, and usually assumes a characteristic position with the good ear downward, resting on the pillow, since this position lessens the severity of the symptoms when he looks at objects around the room. If the patient assumes the upright position, his tendency is always to fall toward the diseased ear, regardless of what position the head may assume. This is an important point in differentiating a diffuse labyrinthitis from disease of the cerebellum. As stated above, to be considered a true diffuse suppurative labyrinthitis, there must be a marked, and usually a total loss of hearing, thus showing that the cochlea also is definitely involved. While any functional tests on the static labyrinth are contraindicated during an active diffuse labyrinthitis, nevertheless it is worthy of note, from a scientific standpoint, that irrigation of the diseased ear, with either hot or cold water, will elicit no response. The well-known triad of symptoms, namely, nystagmus, vertigo, and ataxia, plus a marked loss of hearing, is sufficient to establish the diagnosis.

Every case of diffuse suppurative labyrinthitis passes from the irritative into the latent state, unless meningitis causes death before this has occurred. In other words, a diffuse suppurative labyrinthitis definitely causes a destruction of the labyrinth. When this change from the irritative to the latent stage has occurred, the irritative symptoms entirely disappear and the patient may exhibit no symptoms referable to the labyrinth, except a total loss of hearing. In a certain percentage of cases, until the labyrinth in the other ear has compensated for the loss of its fellow labyrinth, the patient may persist in showing passive (no longer irritative) disturbances in equilibrium, orientation, etc. These symptoms are not spontaneous, and are no longer the result of the diseased labyrinth. From every material aspect this labyrinth is now dead, both as to equilibrium and hearing. Functional examination shows a total loss of hearing, regardless of whether the sound be a high, low or medium pitch. There is absolutely no response to the caloric or rotation test, and there is a negative fistula test. Friesner states that the occurrence of a facial paralysis, in the course of a chronic middle ear suppuration, when the functional tests show a nonfunctionating labyrinth, is positive evidence of a latent labyrinthitis.

The treatment of suppurative labyrinthitis, I believe, is still an open issue. My own tendency is definitely toward conservatism.

During the irritative or manifest stage, I believe no surgery should be instituted, unless there is a series of symptoms indicating impending meningitis, such as increasing fever, headache, vomiting, irritable reflexes and, particularly, increasing cell count of the spinal fluid. The vast majority of manifest cases will pass to the latent stage without complications, if allowed to do so, but when the above mentioned symptoms persist, the labyrinth must be opened and drained. When the disease passes on to the quiescent period, I believe a radical mastoid operation will usually suffice, and should be, wherever possible, the operation of choice. The only other plausible procedure, namely, an opening and drainage of the labyrinth, should, wherever possible, be avoided, as Nature has provided a walling off process somewhere along the tract of that labyrinth which may easily be disturbed during the labyrinthian operation, thus opening up a possible infectious pathway into the meninges; also, the operation itself is extremely difficult, and may easily result in a facial nerve injury, or a surgical mortality in the hands of the average otologist; and, lastly, because of the possibility of a mistaken diagnosis.

In a series of seven cases which have come under my care, all of which recovered, the radical mastoid operation was sufficient in six, while in the seventh a drainage of the labyrinth was required two weeks after the radical operation as the patient exhibited signs of menegical irritation. In one of the first six mentioned the operation was followed by a discharging ear for six months, at the end of which time the cochlea and part of the vestibule were cast out through the ear in the form of a sequestrum, and the ear dried up almost immediately. In another, a secondary radical operation was necessitated, after three months, on account of a persistent profuse discharge, and on reopening the wound the external horizontal canal was completely sloughed off and the superior and posterior canals were uncapped. This case also dried up, almost immediately, following a secondary radical mastoid operation. The remaining four cases had an uneventful recovery. These cases seem to show how skilfully Nature will protect the meninges, if not too strenuously interfered with. I personally feel that a labyrinthian operation on all of these seven cases would have resulted in at least some mortality. In all cases, however, of a labyrinthitis, a radical mastoid should be performed owing to the possibility of the latent condition lighting up at any time and producing a fatal meningitis.

In conclusion, it seems that:

First, a simple classification of labyrinthitis is one of location

with only three classes, as follows: *1.* Perilabyrinthitis. *2.* Localized labyrinthitis. *3.* Diffuse suppurative labyrinthitis.

Second, it is extremely important to do a complete functional examination on all chronic discharging ears.

Third, conservatism, rather than radicalism, seems, to the writer, to be procedure of choice.

145 East 54th Street.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, Inc.

The next annual meeting of the Society will be held in Chicago, Ill., June 8, 9 and 10, 1933, under the presidency of Dr. Joseph C. Beck.

This meeting will follow the opening of the "Century of Progress" Fair in Chicago and will immediately precede the meeting of the American Medical Association in Milwaukee.

The Fellows of the Society will provide the usual excellent scientific program, the energetic Committee on Arrangements, composed of our Chicago members, will live up to its well known reputation for hospitality and the railroads will do their share. We can, therefore, look forward to one of the best and largest annual meetings we have ever held.

As soon as the Committee on Arrangements has decided upon the headquarters for the meeting, notification will be given so that early reservations for accommodations may be made.

Those planning to present papers should notify the Secretary, Dr. Robert L. Loughran, 33 East 63rd street, New York, promptly as the scientific program will be made up at the meeting of the Council on Jan. 7 next.

THE VALUE OF DRY TREATMENT IN ACUTE INFECTIONS OF THE EAR AND MASTOID.*

DR. HUGH B. BLACKWELL, New York.

Prior to the development of the modern mastoid operation and its application by specialists, the general surgeons and family practitioners who were called upon to treat acute abscesses of the middle ear widely held the belief that irrigation of the aural canal in such cases was a dangerous practice and one likely to induce mastoiditis. This belief was the result of years of clinical experience. Such was my own instruction while a medical student.

We are all very careful when washing out the nose of a patient who is suffering with Coryza or a purulent sinus infection and it is well that we are, otherwise the patient would be very apt to develop a middle ear abscess, but are we equally particular about the irrigations which are commonly prescribed for acute middle ear abscess? I have found mothers and even nurses who should know better suspending douche bags four or five feet above the patient's head instead of less than half this distance, the force being intensified by using as a nozzle a glass pipette with a very small opening, the drum and soft tissues of the auditory canal being subject to these forced irrigations with a quart or more of solution at intervals of two hours. Such practice I believe to be faulty and apt to induce mastoiditis, which we wish to avoid. The use of irritating solutions, such as bichloride of mercury and salt, I am glad to say, has been largely discontinued.

It is well known that in some cases, particularly in those of young children and adults, that irrigation of the auditory canal, when the drum is perforated, will force the solution per eustachian tube into the pharynx. Why is it not equally true that in cases of O.M.P.A. without mastoiditis that the aural irrigation would force the pus into the mastoid antrum and produce mastoiditis?

During the early stages of an O.M.P.A. there is, of course, intense congestion in the tympanum, epitympanum and, I believe, the mastoid antrum. This latter structure is where nature makes its last

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stand in defense of mastoid infection. When once the infection passes the antrum, usually the rest of the process becomes rapidly infected. During the second or third week of a mastoid infection, granulations begin to form in the mastoid antrum which more or less interfere with the discharge of pus from the mastoid. In some instances the growth of these granulations completely seals off the mastoid from the middle ear and permits the latter structure to stage a recovery while the mastoid abscess is being formed in the process itself. During the early stages of an O.M.P.A., the congestion and inflammatory exudate thrown out in the region of the antrum is nature's attempt to seal off the middle ear abscess from the mastoid and, while I have no way of definitely proving it, in view of my own clinical experience I feel that there is a distinct danger of breaking down this wall of protective exudate in the antrum region by aural irrigations in cases of straight O.M.P.A., and thereby carrying the infection from the middle ear to the mastoid.

It is supposed that the use of a suction ear douche will preclude this possibility, but does it? In irrigating an ear with perforation of Membrana Tympani, when once the solution leaves the tip of any syringe it is out of our control and just how far it goes before it gets back I do not believe anyone can say. It is conceivable to me that the act of swallowing, crying or contraction of tensor tympani induced by a hot solution might aid in carrying the solution into the mastoid antrum.

When prolonged and frequent irrigations have been used the tissues of the external auditory canal are apt to become hydrated. Under these circumstances the posterior wall will assume a sagging appearance suggestive of an operative indication. In quite a number of such instances I have observed this condition clear up when the irrigations were eliminated and dry treatment substituted. When irrigations are employed and the aural discharge grows less or ceases I think the importance is generally recognized of immediately diminishing the amount and frequency of the irrigations and terminating the irrigations altogether as soon as the discharge stops, as the wet treatment is apt to continue the discharge.

Anatomically speaking, mastoid inflammation and middle ear abscesses differ from those elsewhere in the body in that the avenue of drainage, namely, the external auditory canal, being a fixed bony structure, its lumen remains open except in rare instances, such as cases of exostosis, or when great prolapse or swelling of the soft structures covering it occurs; this is usually produced by external

otitis and not associated with mastoid or middle ear infection. Irrigations of the external auditory canal can only remove pus from that region and possibly from the middle ear in cases of acute middle ear abscess, and I do feel that in the haphazard manner in which aural irrigation is so apt to be practiced that there lies distinct danger of producing the very condition which we wish to avoid, namely, mastoiditis.

I have had a number of cases of mastoiditis which have been previously treated by irrigation and looked like operative cases and, in which, as a last resort, I have discontinued irrigation and applied dry treatment. Many more than half of these cases recovered without having to undergo mastoidectomy. I believe if dry treatment is adhered to from the start more of these patients would be saved from the operating table.

We are, of course, familiar with the beneficial results which follow the use of dry treatment in chronic suppurative mastoiditis and otitis media, also with the use of dry packing in cases of external otitis as against irrigation. In recent years I have extended this practice with good results to cases of myringitis, O.M.C.A. which was not incised, tubal catarrh, and tubo-tympanitis.

The method of dry treatment which I employ in acute middle ear abscess is as follows: After incising the Membrana Tympani, a narrow plain gauze drain is placed down to the drum, the canal being lightly packed. After one-half to three hours this is usually removed, the canal wiped free of clots, usually using a little peroxide, and the drain reinserted. Depending on the amount of discharge, this should be repeated two or three times every twenty-four hours. In some instances a small quantity of boric acid powder is insufflated prior to placing the drain. I believe that the gauze drain, the tip of which is placed against the membrana tympani, has some capillary attraction in securing drainage from the middle ear and mastoid. It follows, therefore, that the otologist must see his patient more frequently than if irrigations were used. When once a proper myringotomy has been performed, I have always felt that secondary incisions were usually of little benefit if the discharge should suddenly diminish or cease and the mastoid tenderness persist. By using a gauze drain I have been able in some of these patients in which the canal was dry to produce a return of the discharge and relief of the mastoid symptoms.

When the patient cannot be seen so frequently and the discharge is profuse, the mother or nurse is instructed to remove the drain after a half hour, instill peroxide in the canal and dry the canal

as well as possible with cotton. This should be repeated at three or four hour intervals.

In the absence of urgent symptoms indicating intra-cranial complications I believe it is reasonably safe to apply this method of treatment for two or three weeks or more before considering the question of mastoid operation.

CONCLUSIONS.

1. I would not care to go on record as opposing irrigations in acute infections of the middle ear and mastoid. I believe, if properly applied, gentle irrigations in combination with dry treatment has definite value in selected cases. I do, however, oppose indiscriminate irrigating in all cases of acute mastoid and ear infections.

2. When using aural irrigations in these conditions our directions should be more explicit to the individual who carries out our instructions. Simply telling the mother or nurse to irrigate the ear at stated intervals is not sufficient.

3. I believe the straight dry treatment employed without irrigations has distinct value if intelligently used in acute infections of the ear and mastoid.

Lastly, I have purposely refrained from quoting particular cases. It suffices to say that I have been using this method for the past three years in treating hospital and private patients, and I have treated upward of several hundred in this manner and the more I employ the straight dry treatment the more I have become convinced of its value in acute infections of the middle ear and mastoid.

2 East 54th Street.

GANGOSA—OCCURRENCE IN A WHITE MAN.

DR. M. C. MYERSON, New York.

Gangosa, also called rhinopharyngitis mutilans, granuloma gangrengosa, kaninloma, may be briefly described as a destructive condition of the palate, nose, and certain exposed skin surfaces of the body. The name Gangosa is derived from the Spanish word gangosser, which means to snuffle. The involvement of the nasal and palatal structures makes a prominent symptom of snufflling speech and breathing. The necrotic process slowly spreads until the extent of destruction is considerable. This paper concerns a case of Gangosa occurring in a white man in a locality where the disease does not ordinarily exist. The patient died as a result of this disease, a circumstance which is unusual.

Historical: Brienl states that Gangosa was first described by a Spanish Commission to the Ladrone Islands in 1828. He also mentions that Dr. Wilson of the Royal Navy, referred to the presence of this disease in the Murray Islands in 1822. Very little was known of this condition until after the Spanish-American War, when medical officers of our Navy were assigned to duty in Guam. With the acquisition of Guam, these officers observed and studied this condition and added materially to our knowledge of it. Among these were Stitt,[13] Bachman,[3] Odell,[11], Angeny,[1] Garrison,[5] Kerr,[8] Johnson and Depping,[7] Kindleberger,[9] Geiger,[6] and Rossiter.[12]

Distribution: The disease is of limited geographical distribution, being confined principally to the Pacific Islands. The disease has been observed, however, in certain parts of Italy. Gangosa is occasionally observed in large seaport towns where sailors who are affected with this condition come ashore.

Etiology: Sex: Brienl[4] states that a majority of those affected are women. Angeny[1] and others believe that males and females are equally affected.

Age: All ages except infancy are prone to the disease. Odell[11] reported the ages of his cases as between 12 months and 84 years. Rossiter[12] reported the condition in a two-year-old child.

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The exact etiology and the method of spread of the disease is not known. Propinquity seems to be an influencing factor; Angeny has recorded the affliction of a mother and four children within a year, the youngest child being eleven years of age. All cases were of the nasopharyngeal type. This would suggest a common source of infection. The disease appears to occur among natives of the lowest classes who live under unsanitary conditions.

Brienl claimed that the cryptococcus mutilans was the causative agent, but this could not be substantiated by inoculation experiments. Some believe it to be a form of tertiary yaws or that it is a sequel of yaws because many of the patients have had yaws.

Odell contended that Gangosa was a form of syphilis, but diligent search has failed to reveal the presence of trepanema pallida in the lesions of this disease. It is further noted that the disease has been common in Guam, where syphilis did not exist. Also antiluetic treatment is of no value. According to Angeny, neither primary nor secondary lues was encountered by the Navy medical officers during a period of twelve years. It has been observed that the females who have Gangosa do not abort, but have a normal pregnancy and bear healthy children.

Gangosa is considered to be a distinct entity by almost all observers. Several writers have predicted that this disease would soon be rare in Guam because of better hygiene and sanitation, and the isolation of those affected. This has proven to be the case. Mink and McLean[10] suggested that flies probably carried the infection from wounds. They also mention the common use of towels, clothing, pipes, cigars, and the absence of dietetic hygiene.

Course: The disease begins as a small nodule which rapidly becomes an ulcer, usually in the midline of the hard palate, but it may begin on the mucosa of the soft palate or the posterior pharyngeal wall. The lesion which begins upon the palate soon gives way to a fistulous opening into the nose. The ulcerative process progresses so that it may destroy the hard and soft palate, the cartilages and bones of the nose, destroying the eye and involving the frontal and ethmoidal regions. In some cases the destruction is so extensive as to include the hard and soft palate, the entire nose, both antra and the orbits. The destructive process spreads in all directions, but more rapidly in the planes parallel to the surface. The bone is lost in the form of large sequestra as a result of a process of slow disintegration. In milder cases the process may be limited to a perforation of the septum, or an ulceration of the soft palate and posterior pharyngeal wall, which results in a scar. The entire front

of the face may be destroyed and present openings with black necrotic bone protruding from the sides. The upper lip usually is spared and in many cases one looks into a cavity whose floor is represented by the tongue. The disease may stop at any stage.

In addition to the lesions of the structures of the head, the extremities and those parts not ordinarily covered by clothing are frequently involved. The ulceration in these regions remains quite superficial, healing in one place and spreading to another. The scars resemble the scars of burns and deformities of the hands and feet may result. There may be contractures or loss of structure or both.

The disease usually lasts a few months to many years. All cases, it is stated, ultimately recover and almost without exception the general health remains good. This was not so in the case of Arrowsmith's patient, who died after a little more than twenty-two months, and my patient, who died twenty-three days after the onset of his illness. Death usually occurs from some intercurrent disease, such as tuberculosis or dysentery. Recovery occurs without treatment. Mink and McLean mention a fulminating form of the disease in children under five years of age, in families where the disease previously existed.

Brienl divided his cases into three groups. The first group included those patients who were ill only a few months, and in whom the disease was localized to the skin of the face and the cartilaginous nasal septum. There was no bone involvement. In the second group the ulceration was farther advanced. Large weeping wounds of the face existed, together with extensive destruction of the bony nose and hard palate. The third group represented those cases in which the nose was entirely destroyed.

Angeny has called attention to the fact that the ulceration is not accompanied by pain and that the individual is not aware of his trouble until the lesion is of considerable size.

Gangosa is extremely rare in the white race. In 1907 Stitt reported the case of a white enlisted man of the Navy who had spent several years in close contact with the natives of Guam. This patient had a fistula through the hard palate which entered his nose. The lesion progressed slowly for five months despite vigorous antiluetic treatment. No organisms could be demonstrated by various staining methods.

In 1921 Arrowsmith[2] reported an interesting case occurring in a white man, in the pages of this journal. His patient was an Italian laborer whom he observed at the Kings County Hospital. Seventeen months before admission he had noticed a hard lump on the

side of his nose which was painless at all times. The left antrum became perforated and the pharynx and nasopharynx were extensively ulcerated. All laboratory tests were negative. The patient died of general debility and inanition.

Case Report: A white Italian laborer was admitted to the hospital in April, 1931. He was the father of two children and had been in this country eleven years, having come here from Milan. A few days before admission he had noticed a spot on his hard palate which became swollen. Within a few days an opening between his palate and his nose appeared. An extremely foul odor was noticed. Both nares were filled with necrotic pus and two perfora-

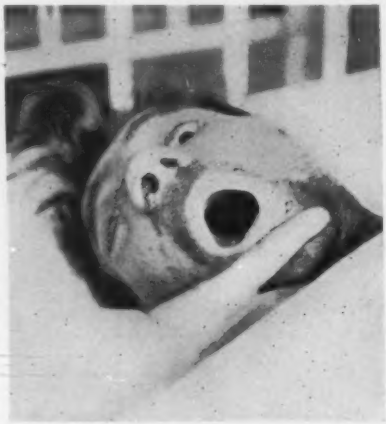

Fig. 1. Photograph showing perforation of hard palate at time of admission.

tions were present in the midline of the hard palate. The uvula was absent. The patient's temperature was 102 degrees and his pulse was weak in quality and of rapid rate. The next day there was an ulceration and a slough on the posterior pharyngeal wall. The nasal septum was absent. Gangosa was thought of and carcinoma and lues were eliminated by appropriate tests and studies. Vigorous antiluetic treatment was tried without avail. On the thirteenth day it became necessary to remove some necrotic bone which was lying loose in the naso-orbital region. An incision made to remove the bone revealed that the nasal process of the maxilla was necrosed and that the orbital plate of the ethmoid was entirely gone. Foul sequestra were present in the nasal cavity and in the ethmoid region the cribriform plate appeared to be involved.

Seventeen days after admission the orbital fat was sloughing, the left eyelids were partly gone and the external nose was beginning to disintegrate. Neurologic examination at this time was negative. The patient became progressively weaker and died nineteen days after admission to the hospital. Dr. Best of the Dermatological staff saw the patient before he died and rendered a diagnosis of Gangosa. Dr. Arrowsmith was of the same opinion when he saw the patient.

Dr. W. W. Hala, who made the pathological examinations in Dr. Arrowsmith's case, rendered the following report on the tissue specimens sent him for examination:

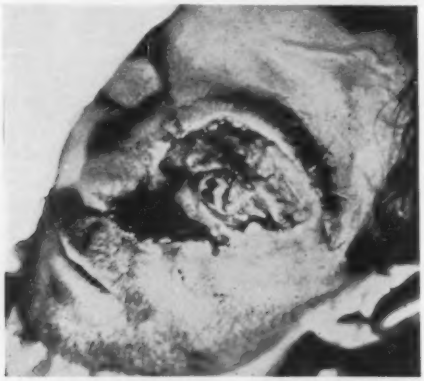

Fig. 2. Photograph eighteen days later showing extensive destruction of nasal and paranasal structures.

"One of the specimens shows skin and subcutaneous tissue with evidence of irregular hypertrophy. The rete pegs reach down rather deeply into the underlying derma. At one side of the section the skin surface is ulcerated. There is a marked predominance of sebaceous glands, probably due to the fact that the specimen was taken from the cheek in the region of the nose, and therefore not a pathological feature. The derma shows infiltration by polyblasts, particularly marked around some of the coil gland ducts and in the vicinity of the sebaceous glands. There is one area situated rather deeply in the subcutaneous tissue, which shows central necrosis. Centrally the structure is absolutely amorphous, but in the middle and peripheral zones of this necrotic area there are plasma cells and occasional epithelioid cells. No giant cells are seen.

The other section consists of markedly necrotic bone tissue, taken from the wall of the maxillary sinus, showing a small limited area which is definitely suppurative and contains, besides the usual granular leukocytic infiltrates, numbers of plasma and small round cells. No giant cells are present.

Diagnosis—Necrosing granuloma."

All laboratory studies were essentially negative. Various studies of the blood and spinal fluid frequently repeated, bacteriological smears and cultures, yielded no information of value.

SUMMARY AND COMMENT.

A case of Gangosa occurring in a white man in Brooklyn, is reported. The disease ran a fulminating course and the patient died twenty-three days after its inception. This is the third case occurring in a white man according to the information of the writer.

The question arises as to whether our patient had a period of incubation of eleven years or more, if the disease had existed in Milan from whence he came to this country.

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12 East 86th Street.

THE TREATMENT OF ALLERGY BASED ON THE CONCEPTION THAT IT IS A POTENTIAL ALKALOSIS.*

DR. ARTHUR M. ALDEN, St. Louis.

The conception that "specific hypersensitivity" is the sole underlying factor in allergy leaves many of the vagaries of this very interesting disease difficult or impossible of explanation.

1. If this is the sole factor, why is it that most allergic individuals show positive skin tests to more than one foreign substance; in fact, many of them show a whole group of sensitivities to widely different types of allergens. *2.* On this basis, how is the factor of heredity correlated with the fact that the parent may be sensitive and give reactions to one type of antigen, while the offspring may show hypersensitivity to widely different types of substances? *3.* Why is that to the same offending substance, one individual will show a rhinorrhea, while exposure to the same substance in another individual produces an urticaria? *4.* Why is it that skin tests are only reliable in about 50 per cent of cases and that after desensitization and apparent clinical cure, many of the reactions still remain positive? *5.* Why is it that in the allergic individual, during other diseases which are accompanied by an upset in the acid-alkali ratio in favor of the acid side, there is often, during this period, a more or less complete remission of the allergic symptoms? *6.* Why is it that diabetes, which is an acid-producing disease, seems to almost confer an immunity to allergy? Jocelyn saw but six cases of asthma in more than 6,000 diabetic patients.

I have no desire to undervalue the importance of the sensitization tests or the therapy based upon the withdrawal of the specific allergy producing agent. These are all of great value and have done much to render more comfortable the existence of these unfortunate allergic individuals. However, it would seem logical to suppose that there is some more fundamental underlying difference in the makeup of the allergic patient as compared with the normal individual. The suggestion by Beckman that the allergic patient is a person whose fundamental body chemistry, either due to an hereditary variation

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in type or to an acquired tendency because of a long continued alkaline nutrition, varies consistently toward the alkaline side, is certainly worthy of some consideration. This hypothesis makes many of the otherwise unexplainable features of this disease much less difficult to understand.

The fact that certain diseases are usually accompanied by an upset in the acid-base ratio in the body fluids has been known for a long time, but only within the last few years, and particularly since the advent of modern physiological chemistry, has this observation been put to practical importance in the treatment of these conditions. Acidosis as the result of diabetes or of depletion of the alkali reserve due to starvation, infections, or vomiting and diarrhea in children, has been carefully studied and an entirely new and very successful form of treatment based upon the replacement of the lost alkali, has yielded brilliant results. On the other hand, alkalosis with its typical pathology—that is, edema, is being also studied by many observers and certain of these studies bid fair to throw light into some of the dark passageways of our understanding of certain otolaryngologic conditions.

The normal processes of metabolism involve a continual production in the cells of acids, both volatile and fixed, which must be promptly disposed of or neutralized. This disposal or neutralization is affected ordinarily, partly by ammonia formed from the deaminization of proteins and amino acids and from the buffer action of the mixtures of phosphates and carbonic acid together with hemoglobin. In certain diseases, this neutralization takes place at the expense of the fixed base in the body cells. Inasmuch as both acids and bases are constantly being utilized and excreted in the maintenance of metabolism, a fairly normal ratio of their intake must also be maintained. This, of course, means that for health a properly balanced nutritional regime from the standpoint of acids and alkalies is a fundamental prerequisite.

The conception that an improper nutritional regime which implies a dietary balance consistently in either one direction or the other may, in time, form a cell and fluid background in the body which predisposes to certain pathological conditions, is only a corollary to what has gone before. In other words, it would seem logical to believe that a patient who has for years lived upon a diet, the metabolic end product of which is largely acid which must be excreted and disposed of at the expense of fixed base in the body cells, would eventually show some clinical results attributable to such a chemical upset. Jarvis, several years ago, published the observation that certain nasal conditions which he then classified under the title of

"Nutritional Disaster Syndrome" were the result of a diet which was consistently heavy in acid-ash foods. He found that by rearrangement of the patient's nutrition with the limitation of these acid-end product foods and the substitution in their place of alkali-ash foods, it was possible to effect a definite change in their symptoms. Beckman, on the other hand, has thought that the majority of allergic patients show a definite acid-base ratio in favor of the alkaline side, and has called them "Potential Alkalotics." Treatment of these patients with acid therapy, coupled with restriction in the intake of alkali-ash foods, has yielded results in cases which could not be controlled by other means.

Until the discovery of the phenomenon of specific hypersensitivity and the elaboration of this later into a working hypothesis to account for all of the manifestations of what we now call allergy, hyperesthetic rhinitis and hay fever were the *bete noire* of the rhinologist. Prior to this time, ambitious nasal surgeons almost ran amuck in their efforts to cure these conditions by operative methods and we are all seeing daily the unfortunate end results of their misplaced surgical zeal. With the advent of this new conception in nasal pathology and the promise that it held forth for the diagnosis and relief for these troublesome conditions, most of us felt that desensitization and removal of the offending allergens offered almost a panacea to these patients.

In the last decade, a voluminous literature has appeared on this subject and today we have the allergist as the most recent addition to the group of medical specialists.

Unfortunately, the treatment of allergy based upon the anaphylactic hypothesis has fallen far short of our expectations. The detection and elimination of the offending allergens in certain cases of food sensitization has yielded many spectacular cures, but the desensitization by protein therapy has given much less certain results and in not a few cases has been the cause of near or actual tragedies.

During the past few years, I have made no effort to treat the cases of allergy which I encountered in my practice, but having made the diagnosis, have referred them to allergists for their therapy. This has resulted almost invariably in the loss of the patient and in many cases the end result was a patient who was dissatisfied with the treatment at the hands of the allergist and displeased at me for having sent him.

With some of these thoughts in mind when Beckman published, in 1929, his new conception of the whole problem, and a treatment that was in accord with it, I at once intended to try it on a few patients. Before I could get it started, however, one of my friends, a

very capable internist, told me that he had treated several patients by this method with little or no results, so I abandoned the thought.

Last fall, as the result of my contact with the men who, under the direction of Dr. Jarvis, are studying this general group of conditions which may be the result of acid-alkali imbalance, I was again prompted to try the acid therapy on some of these allergic patients. The results in a few of these cases, I am reporting today.

Case History No. 1: Miss A. B., an employed girl, age 30 years, was seen on account of headache between the eyes and over the vertex. For the relief of this condition, she had had many nasal operations, including a submucous resection, and later, a double sphenoethmoidectomy; all, however, without relief from her headache. Later she had been tested and given positive skin reactions to more than a dozen foreign proteins. When examined, her nasal mucosa was very pale and at once suggestive of allergy. Smears of the profuse nasal secretion showed more than 25 per cent of the cells present to be eosinophiles. She was given dilute nitrohydrochloric acid in gradually ascending doses and a nutritional regime which was largely on the acid side. At the end of two weeks, her headaches had entirely disappeared. A short time later, however, due to an acute gastric upset, the acid treatment had to be discontinued and the headaches returned. After ten days, she again took up the acid and her diet with prompt relief from her headaches. At the end of three weeks the acid was discontinued, but the diet continued as before. For two months now, she has had no return of the headache.

Case History No. 2: Mrs. H. E., this patient, a married woman, age 46 years, was seen on account of nasal obstruction, occasional attacks of sneezing and rhinorrhea, and paroxysmal headache. All other causes for the headache had apparently been eliminated by a very careful physical examination. When seen the first time, the patient was having no symptoms. A few weeks later, she came to the office with an exacerbation of the headache and her nose now presented quite a different picture than when seen before. The mucosa was pale and water-logged, and smears from the secretion showed 14 per cent of eosinophiles. Based on the supposition that this was an allergic headache, she was given dilute nitrohydrochloric acid, fifteen drops, three times a day after meals and an acid diet. Three weeks' conscientious trial of this treatment produced no relief from the headache. Careful questioning, however, now brought out the fact that for several years she had been taking large quantities of sodium-bicarbonate for a supposed tendency to indigestion. She was advised to discontinue the soda and within five days after this time all her headache disappeared. This was several months ago and when

seen last week, she said that she has had almost complete relief from her pain.

Case History No. 3: F. H., a school boy, age 19 years, seen at the request of his father because of nasal stuffiness and an obstinate dermatitis. His past history indicates that he had fall hay fever at the age of 12 years and said that this cleared up when he omitted feather pillows. He has been treated at some time or other by most of the allergists or skin specialists in St. Louis, and I quote from the records of one of them here:

"His present illness began at 14 years, in the spring or early summer, with typical eczema in the fold of the arms. It spread to the hands, then to the face and neck, but never has been anywhere else. Occasionally this disappeared in summer, but was present all winter. Examination showed a typical allergic dermatitis, localized to the face, neck, hands and forearms. None elsewhere. Physical examination was otherwise negative. Skin tests gave a marked reaction to feathers and ragweed, and a very strong reaction to an extract of house dust collected from his bedroom and living room. Obviously, this is a case of contact dermatitis, in that his skin shows lesions only where it is exposed to the air. That there is something in the dust of his house which causes this lesion seemed evident, not only by the intradermal tests to his dust extract, but also because a patch test with this dust extract was strongly positive. Incidentally, he was drinking a great deal of milk, and because of this excess in his diet, it was restricted. We have never found out what there was in the dust that gave him trouble. We began desensitizing him with his dust, but on a very small dose he became worse. We then did the simple procedure of protecting his hands and face with zinc-oxide ointment against exposure, which made him only slightly more comfortable."

This boy's condition when I examined him was pitiable. His face, neck and arms were covered with long cracks and scratches which were the result of the itching. He had now become so conscious of his condition that he refused to go out at all socially. Examination of the nasal smears showed about 8 per cent of the cells present to be eosinophiles. Based upon this and his previous examinations, the diagnosis of allergy was apparently certain. He was given dilute nitrohydrochloric acid and a diet composed for the most part of acid-ash foods and told to report in two weeks for further observation. At the end of a week, the father called up to say that he was perfectly delighted with the result and that the boy was more comfortable than he had been for several years. At the end of three weeks, the skin condition was very much improved and the nasal obstruction was entirely gone. The dose of acid was increased from 15 to

20 drops, three times a day, and he was given in addition to this a tablespoonful of cod liver oil once a day.

Three weeks later, the dermatitis had almost entirely disappeared and the dose of the acid was reduced to 10 drops, three times a day, which he is still taking. When seen recently, nine weeks later beginning the treatment, the itching has entirely disappeared, it is no longer necessary to use any skin medication, and the patient feels that life is once more worth while living.

Case History No. 4: Mrs. P. M., married woman, age 35 years, referred because of inability to breathe through her nose. The patient says that for four years she has been a mouth-breather. She has had polyps removed many times and has decided that all rhinologists are quacks. She has been in the hands of several allergists and has been found sensitive to wheat, corn, citrous fruits and house dust. For many years, her diet has consisted for the most part of vegetables with some meat. She does not eat sweets because she is afraid of gaining weight.

Examination showed the nasal mucosa to be very pale and waterlogged. The nose was so full of polypi that it was impossible to see the middle turbinates. Smears from the profuse nasal secretion showed a preponderance of eosinophiles. About a dozen polypi were removed at the first sitting which gave the patient more comfort in the way of breathing space. She was given an ephedrine nasal spray, but no change was made in her diet. She returned three weeks later with almost as much obstruction as before and the polyps were again removed At this time, she was given dilute nitrohydrochloric acid, 15 drops, three times a day after meals and her diet changed to one with a preponderance of acid-ash foods.

At the end of two weeks, the patient stated that her nose felt better than it had for several years. A month later, she had had good breathing space and the remaining polypi appeared to be receding; however, at this time she contracted an acute upper respiratory infection and the acid was discontinued. She recovered quickly from the grippe. The diet was continued as before, but she was allowed to go without any acid treatment.

She was seen recently, six weeks since her grippe infection and since the time that the acid was discontinued. Examination of the nose now shows only a very small polyp in the right middle meatus, the left side being entirely clear. The nasal mucosa is pink and practically normal in appearance. Patient volunteers the information that during the last three weeks, she has been able to smell apparently normal for the first time in many years.

Case History No. 5: J. S., school boy, age 8 years, seen because of

asthma and nasal obstruction. He has had an adenoidectomy at the age of three and tonsillectomy and adenoidectomy again at six. He has had asthmatic attacks and nasal obstruction beginning at the age of two-and-a-half. He has been treated by two different allergists and has given positive skin tests to egg yolk, rice, wheat, horse dander and milk products. As a result of the foods that it was necessary to eliminate from his diet, the feeding of this child has been a difficult problem and only a moderate diminution of the symptoms have been the result.

Examination showed a pale undernourished boy whose mucous membranes were typical of allergy. There were no polypi present, but X-rays of the sinuses showed marked cloudiness over each antrum. He was given dilute nitrohydrochloric acid, ten drops three times daily after meals, and after nine days, his nasal obstruction was less marked and he had had only one asthmatic attack. Forced feeding was now instituted, the additions being chocolate and malted milk, and cod liver oil was added to his medication.

Six days later, he suffered another asthmatic attack which was quite severe and lasted several hours. When seen five weeks after this, the mother reported that he had had no more asthma despite the fact that on several occasions he had eaten some of the forbidden foods. Nasal examination now revealed good breathing space through the nose and a mucosa that was red over the ends of the turbinates and the septum. The mother said, a few days ago, that he had gained seven pounds and had had no symptoms for almost three months, whereas before the acid therapy, the longest period between asthmatic attacks was thirteen days.

I have now treated eighteen consecutive cases of allergy on the acidification basis. These were all of the perennial type of the disease and were diagnosed as allergy by the standard methods, history, skin tests and eosinophilia in nasal smears. Three of them show no improvement; however, in one of these I have reason to doubt the co-operation of the patient. For the other two failures, I have no explanation to offer except perhaps my inexperience with this new form of therapy. The other fifteen cases have all been benefited, the improvement varying from an increase in the length of time between attacks to complete relief of all symptoms. I do not know how long this relief will last. The most of them are now off the acid, but all are continuing the dietary schedule which includes an excess of acid-ash foods. I have had no experience with this form of treatment in the seasonal type of the disease, but expect to run a series of these cases during the coming summer. I have seen no bad results attributable to the use of the acid.

Frisco Building.

THE USE OF AVERTIN IN ENDOSCOPY.*

Dr. George R. Brighton, New York.

Avertin as a basal anesthetic is now being used generally in all types of surgery throughout the United States. Reports in the literature are very enthusiastic in recommending its use in otolaryngologic surgery. Some reference has been made to avertin anesthesia in peroral endoscopy and I should like to add our experience at the Bronchoscopic Clinic of the Presbyterian Hospital for the year 1931, more particularly emphasizing its advantage in esophagoscopy.

The drug is chemically a tri-brom ethanol and was first produced by Wilstaetter and Duisberg. The first clinical application was by Butzengeiger in March, 1926. At present, the administration of the drug, at least in our hands, has been entirely the responsibility of a competent anesthetist.

The dosage has been rather carefully worked out and the amount of avertin given varies with the body weight of the patient, although there are other factors, such as general physical makeup, and debilitating and chronic diseases which influence the estimation of the dosage.

The anesthesia is administered by rectum approximately twenty to thirty minutes before the time of operation. It is very rapidly absorbed from the rectum and its effects are manifest in a short time. It produces unconsciousness quickly and quietly. The average time taken in our series to produce restful, quiet sleep has been from five to seven minutes.

The rate and character of the pulse apparently undergo no material change. The muscles of the tongue and jaw become very markedly relaxed and it is most important that a patient during the early stages of avertin narcosis be watched carefully for respiratory obstruction. If obstruction occurs a metal airway should be inserted. Usually the respiratory rate in the case in which a free airway is maintained is somewhat depressed, but the color of the patient remains normal. If respiratory depression or cyanosis occurs in

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cases in which a free airway is maintained, relief can readily be obtained by the inhalation of the carbon dioxide oxygen mixtures.

During the narcosis with avertin there is usually no increase in bronchial or salivary secretions. There has been no nausea or vomiting observed in any of our cases.

In the esophageal cases in this series, thirteen of the group were given 4/10 mils. of magendie hyperdermatically before the endoscopic procedure.

In order to evaluate the success of the anesthesia during the operative procedure we have divided the cases into groups classed as excellent, good and poor.

The cases described as excellent represent complete relaxation with deep narcosis.

Those classed as good showed good relaxation, but a moderate amount of movement on the part of the patient during instrumentation.

Those described as poor showed a considerable amount of struggling and only moderate relaxation.

Out of a group of 22, four were excellent, 16 good and two poor.

It must be remembered that those cases given avertin represent only a small proportion of the total number of endoscopic cases because it was felt that the usual procedure would be distinctly detrimental to the physical and mental well-being of the patient.

Realizing the limitations of any general anesthesia in lung cases such as abscess and bronchiectasis, we have used the drug only on very selected cases when the general condition of the patient would have rendered the operation without anesthesia impossible. It should be remembered, however, that since the cough reflex was definitely abolished for several hours, this type of narcosis is not generally indicated in bronchiectasis and lung abscesses.

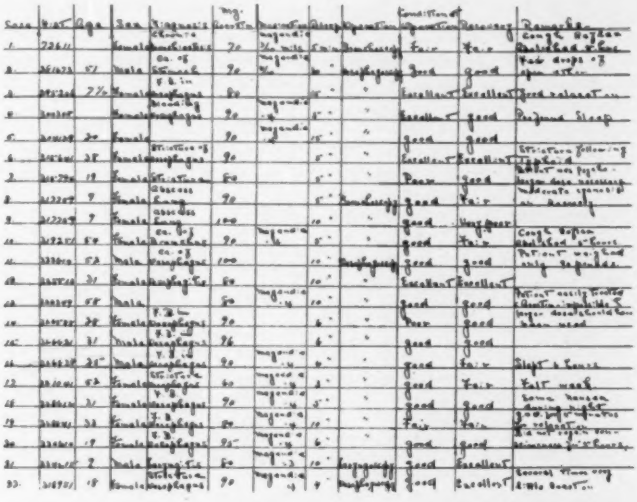
In esophagoscopy one of the greatest difficulties, both to the patient and to the operator, is the involuntary spasm of the cricopharyngeal muscle. In many cases this makes it impossible to introduce the esophagoscope until the patient becomes fatigued and the muscle relaxes. Avertin relaxes the cricopharyngeal muscle very satisfactorily if the proper dosage has been given.

The recovery of the patients we found to be one of the most gratifying features of avertin anesthesia. There is no nausea or vomiting, and the patient generally awakes as if from a natural sleep. He is apt to ask a few drowsy questions and then relapse into another sleep which may last for several hours. Upon questioning, it

is usually found that he retains no recollection of events following the induction. At the Bronchoscopic Clinic we have had no deaths from avertin.

In analyzing the group of cases shown in the appended table, it is interesting to note the preponderance of female patients and the number of cases which were given avertin for foreign bodies in the esophagus.

Out of the series of 22 cases, 16 were of the female sex. It was felt in this group that an anesthetic was distinctly indicated, because of the highly nervous temperament of the patients. In all of the esophageal cases in this group the anesthesia chosen was avertin and was very successful.



The lung cases, three in number, were also given avertin, but we felt that the depression of the cough reflex was great enough not to warrant our continuing this type of anesthesia.

Also, since it is important in cases with foreign bodies in the esophagus to have good relaxation of the patient, we have given avertin to a large proportion of these cases in our clinic. In the present series, eight of the 22 cases represent foreign body problems.

There are two cases of this group of unusual interest. One a child of nine years of age, who had a very severe suppurative pulmonary condition. Several bronchoscopic drainages were necessary.

One was attempted without anesthesia, but the child seemed to do badly. A second one was done, giving a dose of 90 mgm. per kilo per body weight. It was felt that this dose was not quite large enough for the patient. Several weeks later a third bronchoscopy was done, at which time the dose was 100 mgm. per kilo. The bronchoscopic procedure was easily accomplished, but the immediate postoperative reaction was very stormy. After the patient was returned to the ward she became cyanotic; pulse was rapid, irregular and weak. The respirations were very shallow. The blood pressure was 70/50. She was given caffeine, sodium benzoate, but the cyanosis was not relieved. Suddenly, she ceased breathing, the pupils became dilated and there was no corneal reflex. Artificial respiration was given and the patient began to breathe spontaneously. Considerable pus and bloody mucous were aspirated from the throat during this procedure. Two hours later the patient's blood pressure had risen to 98/80 and she was reacting normally, although the cough reflex had not yet returned.

Apparently in this case the dose of avertin was a little too large and the condition was complicated by the inability of the patient to rid herself of the pulmonary secretions.

Another case of interest is that of a child, age 7½ years, who had a jack-stone in her esophagus. She was given 80 mgm. per kilo of body weight. She was asleep in fifteen minutes. The relaxation was complete and Dr. John D. Kernan easily removed the foreign body without trauma to the esophageal mucous membrane. The patient had a peaceful sleep following the procedure and the next morning left the hospital without any recollection of the events of the previous afternoon.

In conclusion, we feel that in avertin we have something which is of inestimable value as an anesthetic in esophagoscopy, but which has been less satisfactory in bronchoscopy.

580 Park Avenue.

THE TREATMENT OF OTO-RHINOLOGICAL CONDITIONS WITH FREE IODINE ADMINISTERED SUBCUTANEOUSLY.

Dr. James R. Bishop, Salisbury, Md.

The value of iodides in promoting absorption of inflammatory exudates has been fully established. Sollman[1] states that "iodides are administered in a variety of obscure conditions to produce 'absorptive' effects."

The coryza of iodidism produced by excessive doses of iodine suggests that controlled medication with this element may be used for therapeutic purposes in treating disturbances of the nasal mucosa. Accordingly, Sternberg[2] employed subcutaneous injections of a 5 per cent solution of sodium iodide containing traces of free iodine—iodomin—in the treatment of nasal and aural conditions of a catarrhal nature. With this treatment he obtained remarkable success, and his results have been confirmed by American observers.

In a series of cases of protracted coryza, vasomotor rhinitis and acute catarrhal otitis media, Rosenberg,[3] in 1932, produced relief of subjective symptoms followed by more gradual objective response by means of iodomin injections. Some cases of catarrhal middle ear conditions responded quickly, after the second or third treatment, and results as a whole were highly gratifying. In cases of coryza and nonspecific rhinitis the outcome was equally satisfactory.

Levine,[4] in 1933, reported twenty cases of pure vasomotor rhinitis and fifty of a mixed type in which favorable results were obtained by iodomin injections. As a rule about six injections were required. In the early stage of common cold and in hay fever his cases also responded successfully. In Levine's opinion, "this treatment proves of value wherever the mucous membrane of the nose is waterlogged and appears to act by regulating the water output of the nose."

In my earlier cases I confined my studies to the use of iodomin for vasomotor rhinitis, hay fever, acute catarrhal otitis media, and the early stage of common cold. The response to treatment was entirely favorable and in accord with the findings by Rosenberg and by Levine.

More recently I have extended the use of iodomin to include pre- and postoperative treatment in rhinology. The effect of preoperative treatment with iodomin, in my experience, improves the surgical field and thereby facilitates the operation. Likewise, postoperative injections shorten convalescence and hasten restitution of the nasal, aural and sinus tissues to normal.

A number of patients with the hypertrophic and hyperplastic types of chronic catarrhal rhinitis who refused operation were notably benefited by a few subcutaneous injections of iodomin. Whether the treatment is given in lieu of surgical measures or in conjunction with them, the best results will be obtained by administering from six to ten ampoules of iodomin at intervals of two days.

In cases of empyema of the maxillary sinus, which as a rule require extended lavages and postoperative treatment, the duration of this treatment is materially lessened by iodomin injections. Usually the antral washings come away clear after the third or fourth injection.

When otitis media is dependent upon underlying lesions existing in the nose or throat, surgical elimination of these foci is definitely indicated, followed postoperatively by ten to fourteen subcutaneous administrations of iodomin.

The following case illustrates the efficacy of iodomin injections postoperatively:

J. S. C., female, age 55 years, married, applied for treatment Dec. 23, 1932, with a history of a chronic discharge from both ears over a period of six months.

On examination, a yellowish purulent discharge was found coming from both ear canals, which were inflamed and reddened. There was no mastoid tenderness. The left maxillary sinus disclosed a discharge issuing from under the left middle turbinate. The other sinuses were clear. Bacteriological examination of the cultures from the sinus and the ear canals showed staphylococci and short-chain streptococci to be predominating. The diagnosis was chronic suppurative otitis media, bilateral, with chronic maxillary sinusitis and ethmoiditis on the left side.

On Dec. 28, ethmoidectomy, antral puncture and drainage, and bilateral myringotomy were performed at the Peninsula General Hospital. After-treatment consisted of lavage of the maxillary sinus at intervals of three days with subcutaneous injections of iodomin. After a period of two weeks, the antral washings returned clean and the left maxillary sinus was clear by transillumination. The discharge from both ears was appreciably decreased. At the

end of one month both ears were perfectly dry. The left drumhead appeared normal, the myringotomy wound having healed completely. The right ear canal was dry, showing no redness or inflammation, but the drumhead remained perforated. Hearing, which had hitherto been reduced to 20 per cent, was now normal in both ears.

The patient, who appeared acutely ill prior to operation, now looked perfectly well. Her appetite improved, and she gained in weight and slept well.

I could cite other causes of a similar nature in which equally favorable results were obtained. It would seem to me from my experience that subcutaneous injections of iodomin are of definite value for combating acute and chronic catarrhal conditions of the nose, throat, sinuses and middle ear, provided proper operative work is done during its administration.

SUMMARY.

My experience confirms the favorable results of Sternberg, Rosenberg and Levine in the treatment of vasomotor rhinitis, acute catarrhal otitis media, the early stage of common cold and hay fever with subcutaneous injections of sodium iodide solution containing a trace of free iodine (iodomin). In addition, I have extended its use and obtained gratifying response in acute and chronic catarrhal conditions of the sinuses and middle ear, where I have found that iodomin injections in conjunction with proper operative measures shorten convalescence considerably and improve the final result.

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Colonial Building.

SOME EXPERIENCES WITH THE NEWER DRUGS.

DR. M. C. JOHNSON, Fort Smith, Ark.

The list of the newer drugs introduced in the past few years in the field of otolaryngology includes among others, euphagin, stryphnon and iodine dusting powder (Sulzberger). Each year sees a startling array of new drugs introduced to the profession. All of them claimed to be superior to the ones in present use. The surgeon, ever hoping for a noteworthy advance, tries the new products, finds the great majority of them overvalued by the maker and returns to his older and time proved stand-bys. However, it is believed that the three drugs mentioned above do have and will continue to have a definite field of usefulness in the field of otolaryngology. However, they, too, in turn may be displaced by newer and more efficacious agents.

Euphagin, developed fairly recently by Haslinger, of Vienna, has been the subject of articles by Laszlo[1] and Robinson.[2] It is dispensed in the form of a tablet and has decided local anesthetic properties. In the article by Laszlo, the use of euphagin in tonsil surgery is confined to the control of postoperative pain. We believe that it has an equally as important use in preoperative anesthesia. It is the custom in some sections of the country, and frowned upon in others, to swab the anterior pillars of the tonsils and posterior pharyngeal reflexes prior to the injection of the procaine solution. Cocaine does this admirably, but there is the danger that with two solutions being used, cocaine and procaine, that through error cocaine might be injected into the tissues with a serious, if not fatal, result. Consequently the use of cocaine in tonsil surgery has been condemned by many. A euphagin tablet, which is not toxic in any degree, may be substituted for the preliminary swabbing of the pharynx with cocaine. Its action is equally as efficient. The pharyngeal reflexes are diminished and the injection with the needle is painless. Any drug which will replace cocaine in any field is worthy of attention. We have also found it useful in securing anesthesia of the throat in the removal by surgical diathermy of the tonsil tags left after previous operation. Laszlo has mentioned its other uses in laryngeal and bronchoscopic work.

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Stryphnon, described by Forschner,[3] is that product obtained before the end step in the synthesis of adrenalin. He advocates its use routinely in cases of tonsillectomy, stating: "We place in the site of operation, after excision, a larger strip of loosely-folded stryphnon gauze to which, on principle, we have recently attached a long silk thread and after a few seconds the wound, which in the meantime has become free of blood, can be inspected as regards a large arterial hemorrhage, which is then arrested by surgical methods. However, many would hesitate to place a vaso-constricting agent in the fossa immediately after removal of the tonsil, preferring that any and all bleeding be manifest and controlled by ligation or suture while the patient is on the table. The objection to vaso-constriction at the time of operation is evidenced by the general trend among otolaryngologists in lessening the amount of adrenalin used in conjunction with the procaine solution used for anesthesia. However, in the generalized oozing from the tissues without evidence of any one vessel being responsible which may occur in the first twenty-four hours after the operation, we have had most excellent results from the use of a tampon of stryphnon gauze inserted between the pillars and held there by their muscular action. As suggested, we have attached a silk string to the tampon, which is anchored to the cheek by means of a small piece of adhesive. The tampon while in place causes the patient no discomfort and the oozing has been controlled. Usually in four hours the tampon is expelled from the fossa. It is then removed and the fossa inspected. It has been uniformly found to be dry. The procedure is not applicable in the control of frank arterial bleeding, but in the cases of pronounced continuous oozing, its use has been most satisfactory and has succeeded where other measures, such as Monsell's solution and others, have failed. In secondary hemorrhages occurring one to six days after the operation, it was found, as stated by Forschner, that the insufflation of powdered stryphnon into the fossa controlled the bleeding with marked success. This, of course, was preceded by the removal of any clot present. It is easy to use and most efficacious.

Iodine dusting powder (Sulzberger), for the treatment of chronic otorrhea, was described by Lederman[4] in 1917, but only recently made available for use of the profession at large. It has also been mentioned by Tsoong.[5] We have had phenomenal success in every case in which it was used. It is realized that this one hundred per cent relief of symptoms will not hold. Any drug which would drv up every chronic running ear, not withstanding any pathology which might be present in the mastoid, is beyond the realms of reason.

Our series of cases have responded uniformly well, a dry ear being obtained in some cases after two treatments, others requiring more. The greater majority of these cases were of long standing, one thirty years, and had failed to respond to other measures, boric and alcohol, removal of tonsils, etc. While this drug cannot be a cure-all, we believe that it offers the best non-surgical treatment of chronic suppurative otitis media in use at the present time and that it will prevent the necessity of doing a radical mastoid operation in many cases.

Diothane, a drug supposed to be superior to cocaine and procaine in its anesthetic properties without any toxic action, is now undergoing clinical investigation. If experience bears out these statements in regard to its properties, it will prove a valuable contribution, as the ideal substitute for cocaine, though long searched for, has not yet appeared.

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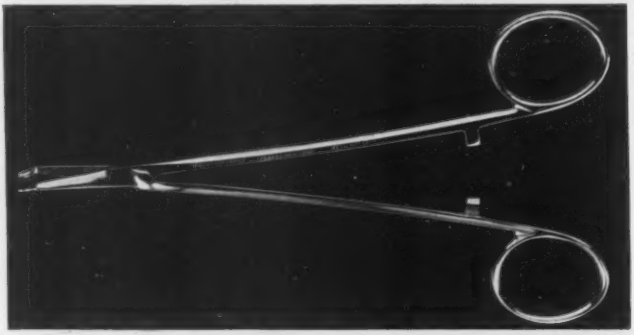
Cooper Clinic Building.

PRESENTATION OF INSTRUMENT—SCISSORS-HEMOSTAT.*

Dr. Arthur Fulton Warren, New York.

This instrument is designed for use with the Coakley slip knot or (single bowknot) in ligating bleeders after a tonsillectomy. It combines scissors with a hemostat or clamp. The clamp at the end of the scissors blades, which constitutes the hemostatic feature of the instrument, is for use in grasping the short end of the slip knot.

The bleeding vessel is first seized by a suitable instrument, such as an Allis clamp. The short end of the slip knot is grasped by means of the jaws of the scissors-hemostat. The loop is passed over the end of the Allis clamp and the loop drawn tight, ligating the vessel below the end of the Allis clamp, which is then removed.



Ordinarily the next step would be to remove the hemostat from the short end of the knot, lay it aside, seize a pair of scissors and cut the long end. By means of this instrument this step may be obviated, for by removing the clamp from the short end of the knot the scissors are in a position to cut the long end immediately. By this means one step in the operation is eliminated and a slight saving of time may be effected. The necessity of removing a clamp from the throat, laying it aside, taking up a pair of scissors and going back into the throat to cut the long end of the knot is avoided.

In addition, we have found this instrument of value in cutting off small tags of tissue about the pillars or elsewhere in the throat or nasopharynx, for in effect we have here guarded scissors with blunt pointed blades which may be used with much less danger than sharp pointed scissors.

50 East Sixty-third Street.

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*Presented before the New York Academy of Medicine, Section of Otolaryngology, May 18, 1932.

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KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Regular Meeting of Feb. 16, 1933.

Dr. Albert N. Lemoine (President), presiding.

Clinical Classification of Mastoiditis. Dr. LaVerne B. Spake.

In surgery of mastoid process we must take into consideration several factors. First, Bacterial, the virulency of the invading organism with its seasonal characteristic. Second, The anatomical pneumatic, diplococci, sclerotic types of mastoid.

I have collected a series of 516 mastoid cases and I find that we can come to certain fairly definite conclusions in regards to a simple classification, taking in consideration the:

(a) The clinical symptoms, time of onset, was paracentesis followed by relief, the length and character of the discharge, the membranum tympanum, its characteristic, with changes. (b) Bacteriological findings. (c) Rotengram. I. Dormant. II. Fulminating. (a) Otitis with meningitis. (b) Hemorrhagic. (c) Influenza. III. Progressive. IV. Atypical. (a) Infantile. (b) Tubercular. V. Chronic sclerotic. (a) Cholesteatoma. 1. Wet. 2. Dry. (b) C. P. O. M. I. Marginal—perforating. 2. Central.

I. The dormant type, the strepmucous type as described by Ruttin. The initial symptoms may be overlooked, spontaneous rupture of drum, followed by a dormant stage of weeks duration, with no pain, tinnitus is present, slight deafness, drum appears grayish and thickened, may be 50 times its normal size, no transparency, short process obliterated; no improvement in hearing on inflation, the discharge at onset may be profuse, with mucoid character, then purulent, the amount subsiding and may be almost negligible; bony involvement is early, with cavity formation. In our series we find from two to four cases each year.

Man forty-five years of age, onset weeks ago with acute earache, spontaneous rupture with discharge for two weeks, present complaint dull headache, sleeplessness, tinnitus, pain and swelling over tip of mastoid. At operation the mastoid process was one large cavity M. M. edematous and completely filling the mastoid, with only few drops of pus present.

II. Fulminating. (a) Otitis with meningitis is that type with sudden onset, acute severe earache, spontaneous rupture or early incision, meningitis and death. Surgery of mastoid may or may not be indicated. I feel that is probably a useless procedure unless there be definite localizing signs in petrous pyramid or labyrinth.

(b) Hemorrhagic type, of fulminating mastoiditis, is due to strephemolyticus.

The initial symptoms, high temperature, severe earache, not relieved by spontaneous rupture or incision. The drum is red, bulging and pulsating; obliterating of short and long process of malleus deafness. The amount of discharge is profuse, may count 20 drops per minute, the type of discharge is always hemorrhagic. The clinical picture is a stormy one which does not subside. Continued high temperature, 105° to 106°. These cases should be operated after the first week and the findings are not the same as is found in the progressive tube, operated too early. The entire mastoid intracellular structure is hemorrhagic with M. M. Swollen, thick and intracellar structure bleeding with each procedure, the cell walls are not destroyed, cells are filled with blood or serum. On operation when the cortex is exposed and thin, the blood filling the cells will appear blue.

Sinus thrombosis is common complication.

Roentgenogram, no intracellular destruction, must be differentiated from progressive type and the otologist must make the final decision.

A scarlet fever otitis can be classified with hemorrhagic type, plus early destruction of membranum tympanum.

(c) Influenza, due influenza bacillus. The initial symptom high temperature, severe earache. Symptoms are slowly relieved by rupture or incision. The drum from the initial symptom is bulging, slightly red, but more grayish, complete obliteration of all land marks with marked sagging of postsuperior canal wall, profuse mucopurulent discharge, changing to purulent. The clinical picture is stormy, but not as severe as hemorrhagic type, leukocyte count is low compared to hemorrhagic type and possibility of mastoid involvement are not great.

Man fifty-five, influenza three weeks ago with A. O, recurrence of mastoid pain, headache, restlessness at night with facial paralysis, on operation found a hemorrhagic type mastoid cavity, no postoperative, temperature, but recurrence of pain at night on the fifth postoperative day. A spinal puncture was repeated with 7000 cell count, with free pus influenza bacillus, repeated spinal puncture with Kerrison operation for meningitis. Case recovered.

III. Progressive. The initial symptoms severe with relief by incision of drum. The ear drum has a sausage like bulging of post quadrant, dilated blood vessel across drum, center of which is bloodless due to increased intratympanum pressure, slight deafness, pulsating, purulent secretion, which changes to a mucopurulent, subsiding and recover, or have recurrence of initial symptoms. plus a difference in type and amount of discharge, apin over mastoid process with edema. The most common sign, sagging of posterior canal wall, pain extending to attachment of sternocleidomastoid muscle, edema and etc. Perforation may occur through cortex, with relief of pain temporarily.

Atypical Type: I. Infantile: In new born we must not overlook an otitis due to foreign body (amnoitic fluid). S. J. Crowe, Archives, August, 1930, reports two cases which demonstrates the persistence of mesenchyme in M. E. and mastoid antrum in person over fifty years.

The cholera-infantum syndrome is conspicuous by its absence in our cases. I had one five months' old infant with all the classical symptom, the drum intact, sagging canal, land mark gone. The patient was referred to a pediatrician for confirmation of my diagnosis; he overlooked the ear condition, treated the dehydration and general treatment, and infant recovered.

I have had a number of classical mastoiditis cases in infancy, most of them of perforated type, may be so-called primary type, but we always have middle ear signs; the youngest case was four months.

T. B. otitis secondary to a pulmonary tuberculosis. The drum perforation are two or more, in same quadrant of the ear drum, with a bridge of tissue between the perforation, as the perforation is caused by a tubercule, which are perivascular and might break down and cause perforation. Painless type discharge may be profuse, tubercular. We find tuberculosis more often in children; operation is best in inactive stage; sequestra of bone is very frequently found.

Chronic Purulent Otitis Media: I. Non-dangerous type. Central perforation we use the iodine treatment. Boric acid and alcohol. Ultra-violet, symptoms of Eustachian tube treatment. Development of acute mastoiditis symptoms with type I, II and III infection, then we feel that mastoidectomy is indicated.

II. Marginal perforation in chronic suppuration without mastoid symptoms and absence of cholesteatoma, the case is kept under observation. Cholesteatoma are primary in suture of skull. Secondary in C. P. O. M. with marginal perforation.

Marginal perforation the epithelium grows into inner wall of middle ear, taking connective tissue with it, part desquamate and have collection of cholestin and formation of cholesteatoma.

Two type, dry can be treated locally, never irrigate, use alcohol for cleaning.

Wet type, irrigate with attic cannula. Radical operation or modified, if chain of osside are intact.

DISCUSSION.

DR. HOMER A. BEAL: I did not have an opportunity to read Doctor Spake's paper before the discussion. It is difficult to carry in mind the entire classification that he has given. I do not think of any such classification in routinely taking care of cases. This is not meant as a criticism of his work at classification.

What are the possibilities anatomically in the mastoid bone? We have different types of bones. 1. Pneumatic type, 2. diploetic type, and 3. sclerotic type where embryonal fluid produces sclerosis early in life and later develops into sclerotic mastoid.

Instead of bearing a classification in mind, I think what sort of an epidemic we are in when having otitis. Those I have had lately are of streptococcic type. Right now we would not need a classification. I have not had the vascular type during this season. Practically all have had frank pus in the cells or granulations and are not bloody as in the vascular type. Intracranial complications come early with the vascular type. We must always think of complications with an acute exacerbation of an old chronic case. This makes easy classification.

I recently had a patient, a girl thirteen years old, who had a stiff neck, suppurating ear and lumbar puncture 5,000 cells with no bacteria in the spinal fluid. Operated and after fourth day the girl is improving. At the time of the operation no break was seen in the dura. There were probably some portals of entry in the epitympanum. Did not do a radical operation in this case.

Lately I have had a few complications in the veins. Typical lateral sinus cases with chills. In one case the patient had a positive blood culture, but we found no clot in the vein of either side.

DR. CHARLES W. COLE: This classification is a little more elaborate than I have studied, but taken as a whole, I think it is a wonderful classification. His resume of symptoms is well covered.

I think we find, especially in the country, that we have to depend on our own judgment. We do not have a chance to call in consultants, so we must develop a certain amount of faith in our own ideas.

In the last two or three years I have had less mastoids to deal with than formerly. Possibly some of the mastoids I had before were due to my method of treating running ears. Recently I have treated ears with dry wicks, first incising the drum for sufficient amount of drainage. Most mastoids are due to a blocking of the antrum by the aditus. If we get sufficient drainage, we have less trouble in the mastoid cells. I have always made it a practice to test the strain of bugs we have and go as much as possible on X-ray findings. I do not depend as much on blood count as on the pulse and general phases of the patient.

Many mastoids in the past ten or fifteen years have been operated far too early. With treatment we could have warded off many of these cases. I recently had an interesting streptococcic case. The drum was affected following flu. The patient is forty-two years old and had had a running ear ten days before I saw him. I enlarged the incision. The patient was having pain in the other ear but no indication for opening the drum. I took the precaution of opening it anyway. He had an enormous amount of swelling around the ear, but this soon subsided with ice packs.

I enjoyed Doctor Spake's paper very much and think it is timely at this season of the year.

DR. ALVIN J. LORIE: I want to compliment Doctor Spake on his classical classification. It shows a lot of work and is very inclusive.

The most important item we have to deal with is X-ray. I know of nothing that does more to sell a mastoid operation to the patient and does less good to the doctor than an X-ray picture. We have worked with an X-ray expert here until he is so confused he will not give a diagnosis from his pictures. We have taken cases which he has diagnosed a mastoiditis and shown him that such was not the case. We have also taken cases where he stated there was no mastoiditis and invited him to watch the operation and see the extent of the necrosis.

In the histo-pathology of hemorrhagic mastoiditis the necrosis of the bone is due to thrombosis of small veins obstructing the flow of blood which gives nutrition to the bone.

Types of mastoiditis: 1. Coalescing type, and 2. Hemorrhagic type. Streptococcus mucosa is pneumococcus according to American classification. Europeans call it strep mucosa.

DR. VIRGIL W. MCCARTY: Lorie has taken some of my thunder. Remove from indications for operation the X-ray examination. We cannot make a consistent diagnosis with X-ray.

As to length of time from the original ear discharge to the operation, I suggest to Doctor Beal that ten days is much too early.

Doctor Spake's classification is sincere and earnest. It is an effort to try to find when to operate on a given type of mastoiditis. I think in his next five hundred cases he will simplify it considerably. It is hard to make such clear-cut distinctions.

DR. GILLILAND: I like to classify mastoiditis into the acute and chronic and then use my own judgment in each individual case when to operate and what type of operation to do. I have found that an influenza otitis media differs somewhat from the staph and strep infections in that it comes on suddenly without any evidence of inflammatory changes in the nose and throat and an immediate myringotomy is indicated. However, I have not found that influenzal infections in the ear and mastoid require mastoidectomy any more frequently than do other types of infection.

There is an interesting observation I have made in a few cases of acute otitis media with mastoid involvement in children, and that is that in a few days following onset of suppuration of the ears the patient will have a chill, followed by a rapid rise in temperature. The temperature will subside in a few hours, to be followed the next day by another chill and rise in temperature. This will be repeated for several days. The white blood count will run anywhere from 15,000 to 30,000, the blood culture will be negative, the spinal puncture negative. The roentgenogram of the mastoids will be cloudy, but no cell wall destruction. The mastoids will be somewhat tender, the general physical examination, including a careful chest examination, will be negative, but a roentgenogram of the chest will show a small area of broncho-pneumonia which has been causing all the symptoms. Under appropriate treatment the ear and lung condition will clear up within a couple of weeks. The roentgenogram is the only way that an accurate diagnosis can be made in a case like this.

DR. THOMASON: I never say anything that is worth while. I never heard of one man having so many mastoid cases. I have been practicing thirty-five years and have not had that many. I believe as Doctor Cole that some of us get into them a little early. If you let mastoids alone and incise the drum, Doctor Nature will take care of them.

I had a lady sixty years old down with the flu. There was an ear involvement and I found a bulging drum. Incised and out came blood and pus. She had a tender mastoid, some edema, chills and fever and sweats. Some of you young fellows would have operated. We gave her aspirin and argyrol for her nose and hot applications. She is up, around and happy. I told her if she had another chill I would take her to the hospital, so she got well. Bluffed her out of it. This was three weeks ago and although she cannot hear perfectly, she feels well, has no fever and is getting along all right.

X-ray looks pretty good to me and is a selling point. Will continue to take X-rays.

Many patients are getting well without operations. An immediate operation is seldom necessary. Patient will accommodate himself to infection if given time.

DR. W. L. POST: Want to talk in behalf of operation. I would rather operate twice too soon than once not soon enough.

DR. AMBROSE EUBANK: I heartily agree with Dr. McCarty that you make more mistakes operating too soon than too late.

DR. L. B. SPAKE (closing): In classinfying surgical mastoiditis we have a number of conditions to consider. First, we must differentiate a hemorrhagic mastoid from progressive mastoiditis at the different stages.

If in the fulminating type the infection is in the mucous membrane lining the cells and not the bony intracellular structure, then bone necrosis is always late.

The object of this paper primarily was to find out from my own experience the mortality rate.

In this series of cases we found that 90 per cent of the cases which came to operation were cases that the drum ruptured spontaneously.

I believe that all cases should be X-rayed. First, to find the type of mastoid; second, the location of the sigmoid sinus; and third, evidence of intracellular destruction, cavity formations or cloudiness.

We must not overlook the factor of time from the onset to the recurrence of symptoms, the type and character of the discharge and the appearance of the ear drum.

Foreign Body in the Bronchus—Case Report. Dr. W. L. Post.

In presenting this case of a peanut in the lung, I merely want to emphasize the unusual presenting symptom of emphysema of the neck which with X-ray was demonstrated to come from emphysema of the mediastinum. We have the history of a child two and one-half years of age developing fever of two degrees, a severe coughing spell and a swelling of the neck four and one-half days after choking on a mouthful of peanuts. During this time there had been only a slight coughing which the parents had attributed to a cold, and the choking on peanuts had been forgotten.

The physician first called found a markedly hyperresonant left lung without breath sounds and coarse rales and increased breath sounds in the right lung. The X-ray shows the heart markedly displaced to the right with a greatly distended left lung and generalized early bronchial pneumonia throughout the lobes of the right lung.

On an attempt to remove the foreign body, a large quantity of pus was found coming from the right main bronchus—so much so that at no time could the peanut be kept in view long enough to be grasped without being covered by the pus. Bronchoscopy was stopped, as the child was becoming tired. A few hours later there was no difficulty in grasping and removing a full half of a large-sized peanut.

Unfortunately, as was to be expected, we eventually lost the child with pneumonia. Our opinion is that there was a body lodged first in the right main bronchus. Four and one-half days after inspiration and at the time of the onset of symptoms which caused calling of physician, the body had jumped from the right main bronchus into the left. At no time was I able to see any ulcerative process in the trachea or bronchial tree. In no other way can we explain the presence of air in the mediastinum.

DISCUSSION.

DR. HOMER A. BEAL: I had a case very much like this who died this morning. My patient had foreign body about ten hours. Results seem to be the same, no matter how long the peanut has been in the lung before removal. Removal of the foreign body was easy, but patient had emphysema of that side of the chest. Tracheotomy was not resorted to until three days after removal of foreign body. Then three bronchoscopies had to be done to remove thick, tenacious secretion from the lungs. This had to be removed with forceps.

Did post-mortem this morning and the main finding was pus below the site of the foreign body along the right main bronchus with ulceration along the trachea. Peanut oil has peculiar effect on lungs in developing tenacious secretion and is very toxic.

DR. PAUL LUX: Emphysema does accompany foreign bodies in the esophagus.

DR. POST (closing): In my case I think the foreign body had originally been in the right lung and jumped to the left. That is the only way to explain pneumonia in the right lung when the foreign body was in the left. The ulceration must have been in the right main bronchus. This is the only reason for emphysema of the mediastinum.

Infection of Sphenoidal Sinus with Intracranial Complications—Case Report. Dr. Evan S. Connell.

W. E. C., male, 32 years of age, was admitted to St. Luke's Hospital, April 10, 1930. He had suffered with headache for about ten days. The day of admission the patient had a fainting spell, losing consciousness and falling to the floor. Vomited several times after regaining consciousness. Vomiting was not of the projectile type. Since then has had pain which he says originates back of his eyes, near the center of his skull, increasing in intensity with an expanding sensation as though the skull would burst.

Physical examination on the day of admission was negative. All reflexes normal. Temperature 98.2, pulse 70, respiration 16. A tentative diagnosis of hysteria was made by the attending physician. April 12th, the following laboratory report was received: Blood, Wassermann negative. Spinal fluid: Culture negative, gold negative, Wassermann negative. Leucocyte count 17,600. I was called in consulation this date. Roentgenogram report of sinuses was as follows: "Normal aeration of all sinuses except the left sphenoid, which appears quite dense." I decided to drain the left sphenoid because of the following factors: 1. Negative physical examination. 2. Roentgenogram findings. 3. Leucocytosis. 4. Persistent headache originating in the region of the posterior sinus group. 5. Temperature ranging from 98.2° to 101°, and of an irregular type.

Drainage of the left sphenoidal sinus April 15th. A small amount of thick, mucopurulent material was found in the sinus. Patient was taken from the operating table to the X-ray department. Roentgenologist reported: "Further studies of the skull, with special reference to the sphenoidal sinus taken after operation, shows a clearing of the left sphenoidal cell." April 16th, headache relieved. April 17th, patient developed a left hemiplegia, complete. Doctor Gibson was called in consultation and made a probable diagnosis of thrombosis of the internal capsule.

The following notes are taken from the progress record of the hospital chart:

April 19: General condition not so good. More stupid.

April 22: General condition improved, especially as to the mental condition. No movement of the left side.

April 23: Mentally normal. Wide awake and active. Ankle clonus more marked on the left side. No voluntary movement.

April 24: Tendon reflexes more active, left side. Face more symmetrical, and on smiling spontaneously, left side of the face moves very well.

April 27: Complains of a numb feeling in the left arm and leg. Can move leg slightly. Cannot grip with the left hand. Discharged from the hospital this date.

There was a gradual improvement and complete recovery of function of the left side. Following operation on this patient, the headaches were immediately relieved. Temperature and leucocyte count gradually returned to normal. It is my opinion that this patient developed an encephalitis, secondary to infection, of the left sphenoidal sinus.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of January 18, 1933.

(*Continued from April issue,* THE LARYNGOSCOPE.)

DR. A. B. DUEL: This program, seriously undertaken by the Oto-Laryngological Section of the Academy of Medicine, is like a revival of "The Mikado" at Radio City. It's nice to hear the old familiar songs again under new auspices.

I have enjoyed these papers and the able discussion of them. For me to attempt a summary of the subject would be like the old man who, after hearing a good trite story well told, breaks in with—"Well, you got the point all right, but this is the way my grandfather used to tell it."

As Dr. Atkins pointed out, there are two distinct types of acute mastoiditis: (a) The usual suppurative type, developing from direct extension of mucosa and submucosa. The infection travels from the nasopharynx along the Eustachian tubes to the middle ear, antrum and mastoid cells. (b) Thrombotic: hemorrhagic type. The venous channels are thrombosed and subsequently break down. This process may skip the middle ear or involve it so slightly as to give no gross clinical evidence. The mastoid cells may become thoroughly involved long before there is any clinical evidence in the middle ear.

In a small minority of cases the diagnosis is difficult to the most experienced observer, fortified by all the available assistance from the clinical and x-ray laboratories.

The factors which enter into these problems have been clearly pointed out, both in the papers and in the discussions by those who have preceded me. Suppose we were to admit—for the argument's sake at least—a point which I firmly believe and with which I am sure many of you also concur, namely, that every case of suppurative middle ear infection is accompanied by an inflammation—either mild, moderate or severe—of some or all of the adjoining mastoid cells. We would then be in a position to say: "Yes, this is a mastoiditis; is it an operative case?"

What would determine this? Often local signs alone, without any constitutional manifestations; often constitutional manifestations without any local signs. The nature of the infecting organism in either of these instances may determine the question of operation. The blood count may decide the question. The x-ray picture may solve the problem.

In a doubtful case, in an infant where movements of the patient may prevent a satisfactory picture, either operate or give an anesthetic in order to get a satisfactory picture. Don't hesitate. In a hospital with proper facilities it is possible to get a determining evidence from the wet plates without stopping the anesthesia. In the face of urgent local or constitutional symptoms, I would disregard a doubtful roentgenogram. In my experience this seldom happens. The picture is more likely to be reversed—i.e., either definitely for or against operation. All this applies to acute cases in the first two weeks.

What of the case that goes on with a profuse discharge for a longer period, having given in this time no alarming symptoms either locally or constitutionally? Nothing has called for x-ray or blood count; the organism is not "vicious." X-rays show pus and granulations throughout the mastoid cells. In my opinion, such a case should be operated. Suppose it should recover in six, eight or twelve weeks? The hearing would be much impaired permanently, whereas if operated at once the hearing would be preserved. There is also a chance during this period that some unforeseen complication may occur. Even without this, the conservation of hearing warrants the operation in such a case.

I have arrived at an age when catching trains "by an eyebrow" instead of leisurely "getting aboard" ahead of time no longer appeals to me. Just as I no longer have a spirit of adventure and do not enjoy taking needless risks. It is all right to fearlessly approach unavoidable dangers, but I don't deliberately go out looking for trouble any more. Enough turns up unsought.

I therefore prefer to operate early on a case which is full of potential dangers when I feel quite safe regarding the outcome rather than to wait for some untoward development to force me into a hurried effort to avert a catastrophe, with all the inevitable uncertainty and anxiety as to the result. I much prefer, on a slippery road, to drive my car so slowly that there is no danger of skidding rather than to drive at such a pace that I am constantly on the threshold of a skid. I may know what to do when the car skids, but I prefer not to try it. The most skilful driver often comes a cropper.

And so I feel that my tendency to operate early on a doubtful case should be regarded as a conservative rather than a radical attitude.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Meeting of February 15, 1933.

Symposium on the Larynx.

(a) **Practical Anatomical Considerations of the Larynx.** Dr. John M. Loré.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(b) **The Contact Ulcer — Etiology and Treatment.** Dr. Chevalier L. Jackson.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(c) **Differential Diagnosis in Diseases of the Larynx.** Dr. John D. Kernan.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION OF DR. LORE'S PAPER.

QUESTION: What effect would the ligation of the superior and inferior thyroid arteries have upon the gland?

DR. LORE: For laryngeal hemorrhage, neither the superior nor inferior arteries would be ligated, but the laryngeal branches coming from them would be the ones to be tied.

QUESTION: What is meant by the adventitious cord?

DR. LORE: That is nothing more nor less than a regeneration of epithelium over scar tissue which replaces the tissue removed. In the laryngofissure operation, the tissue removed is replaced by fibrous tissue, and this is eventually recovered by epithelium. In the case where the cord was stripped or the mucous membrane removed, regeneration of the mucous membrane took place in much less time than in the cases that have had a laryngofissure operation.

DR. J. D. KERNAN: In what case have you used avulsion of the cords?

DR. LORE: Cases in which it has been tried are hyperkeratotic papilloma, which is to be considered as a precancerous lesion; polypoid degeneration of the cords, and multiple papillomata. In the case of hyperkeratotic papilloma, the patient was a lawyer, who had to give up his practice because of his voice. After complete removal of both cords there was a regeneration of tissue, so that now one can hardly distinguish between normal cords and what he has now. He has returned to the practice of law.

In the case of polypoid degeneration of the cords, I had two sisters, one a school teacher. The school teacher had to give up teaching because of the voice condition. In both cases removal of tissue from both cords extending from the anterior commissure to the vocal processes was tried with startling results. In a few weeks both cords appeared to be normal and the voice, which had been very hoarse, was as near normal as one would care to have it. The school teacher is now back in school teaching.

The next phase of the subject being studied is its applicability to cases of bowed cords in which there is incomplete approximation of the two cords. I am of the opinion that in these cases, if the edges of the cords are removed that in regeneration of this mucous membrane, the cords will become tense and thereby permit complete regeneration.

DR. V. C. MYERSON: Is it not true that stripping of the mucosa is not replaced by an adventitious band, as suggested by Dr. Loré but rather by a process of epithelization? This takes from six to twelve weeks. A recent experience that is of value in relation to Dr. Loré's work may be cited: While removing a very small vocal nodule from the cord, the mucous membrane of the cord came away with the nodule. Nothing was thought of this, but twenty-four hours later there was a large brownish mass protruding from the edge. Close inspection proved that this was the thyroarytenoideus muscle which had herniated through the torn mucosa. The muscle was gradually forced back into place by the epithelium which grew in at the anterior and posterior ends. Examination every few days revealed a shortening and narrowing of the muscle mass. This completely disappeared at the end of six weeks.

DR. NATHAN SETTEL: Will you please explain why the thyroarytenoideus externus muscle, forming the outer wall of the ventricle of the larynx, and because of its attachment to the thyroid cartilage anteriorly and the arytenoid posteriorly, shortens the anteroposterior distance of the glottis, is also classified as an adductor of the vocal cords? What is its specific action as an adductor?

DR. LEOPOLD GLUSHAK: Do you do the stripping of the cord under direct or indirect laryngoscopy?

DR. W. J. GREENFIELD: I wish to ask Dr. Loré who, when and how was the discovery made of the innervation of the oblique arytenoid muscle through the internal branch of the superior laryngeal nerve.

DISCUSSION OF DR. JACKSON'S PAPER.

DR. C. J. IMPERATORI: I understand that Dr. Chevalier Jackson has had 250 similar cases during a period of 40 years. I have seen but three cases. I recently saw two of these, and one of them has given me considerable concern.

The patient was one who did a great deal of talking but did not smoke or drink. He is a man of 50 years, in very good physical condition, and his past history has no bearing on his present condition.

While driving his car in the country, he experienced a sharp pain in his throat and thought that he had aspirated a bit of sand or grit, or something foreign. I saw him about two weeks later and at that time there was a small bleb-like formation located on the edge of the vocal cord, posterior to the vocal process. There was a question whether or not this was tubercular in nature. Roentgenogram of the chest, repeated temperature readings, careful observations of the lesion over a period of three months, seemed to prove that this was not tubercular. There was no sputum at all. The Wassermann was negative.

He was seen in consultation by Dr. Coakley and it was considered that it was possibly a papilloma and also the remote possibility that it might be a malignant growth. It was decided to remove it and submit it to microscopic examination. The appearance of the growth, to me, at this time, was that of a granuloma. Under local anesthesia the granuloma which at this time was about 5 mm. in diameter, was removed, and microscopic examination proved that it was granulomatous in origin. No lesion of the arytenoid cartilage could be seen at the site where the granuloma was removed.

After a period of a few weeks, there was a recurrence of the granuloma and an ulceration on the opposite cord, that the granuloma fitted into. The ulceration was about 3 mm. in diameter.

He was seen by Dr. Chevalier Jackson at this time and the diagnosis was given as that of a contact ulcer. He was again Roentgenogrammed and put through the usual course of examination, but with negative findings, excepting a confirmation that this was a contact ulcer. Absolute vocal rest was started and he continued using the pad and pencil for about six months. In the meantime the lesions would change almost from week to week.

He was again seen by Dr. Chevalier Jackson and it was decided that when these granulomas or ulcerations appeared, that it was better to excise them. This was done and apparently with exceedingly good results. The method of procedure was to excise the growth or ulceration, using a cup type of forceps and nipping a small section of the surface cartilage. Within a week to 10 days healing took place. At present, after a period of three months, one side is entirely healed and the other is rapidly healing.

A second case was seen and, profiting by my experience with the first case, I excised the growth, using the method as described, and after a period of five months he remains entirely healed and apparently cured.

DR. H. M. SCHEER: In many of the slides shown by Dr. Jackson the ulceration appears to have a hypertrophic appearance. Would he give a brief description of the appearance of these ulcerations from their onset, and also tell us how frequently he has found it necessary to do the type of removal that Dr. Imperatori has done?

DR. V. C. MYERSON: Although Dr. Jackson has stressed vocal abuse in the consideration of the etiology of contact ulcer, the etiology is not definite to me. That the benign lesions occurring at the junction of the anterior and middle third of the vocal cords are most frequently due to vocal abuse is well established. If the posterior lesions which are being discussed tonight are due to vocal abuse, why is it that we do not have various types of polyps, vocal nodules and fibromata posteriorly? I would like to ask whether trauma has been ruled out in the cases presented? Also whether the bacteriology of these lesions has been considered?

DR. M. J. MANDELBAUM: Would Dr. Jackson consider as a possible etiological factor of contact ulcer, a functional imbalance of the arytenoids caused by inco-ordinated action of the muscles of the vocal cords?

Assuming, for instance, that the muscles of one cord are slightly weaker than the other or, vice versa, the muscles of one cord more spastic than the other, cannot the impact of the more rigid against the relaxed side result in an abrasion? Thus, by the constant rubbing, could this abrasion not deepen to an ulcer of one or both sides?

In the presence of the pathologic bacterial flora normally inhabiting the throat, it is easy to conceive that the mechanically produced abrasion becomes a locus resistentiae minoris permitting secondary infection.

Such a chronic irritative process, by means of proliferative pathology of the cornified epithelium, could produce a prominence on one cord, which, by constant contact, eventually cause a cupping or depression of the corresponding area on the opposite cord.

Regarding the bacteriology, I believe I had in mind a thought similar to Dr. Myerson's, but I think he will agree that the bacterial etiological factor is most likely to be a superimposed causative factor.

The opposite, however, can be conceived, that an infection of the cord might take place and the mechanical factors mentioned above become the secondary factors. Inasmuch as the true etiology is so uncertain, I present this theory for consideration.

DR. JOSEPH KRIMSKY: In reference to the possible cause of this granulomatous condition, I wonder whether cultures from scrapings would not produce the same condition?

DR. M. S. LLOYD: When I was studying pathology I happened to see several of these ulcers on the vocal cords at autopsy. The pathologist explained that if a membranous surface bears an exudative lesion and is in apposition with another membrane, the surface of the second may develop a lesion of the same kind. In that case the opposing surfaces tend to adhere and if they are at rest they will form strong fibrous adhesions and heal. On the other hand, he showed cases where rest of the two opposing surfaces is impossible. In these cases healing is impossible. The end-result in these two instances is exemplified in pleural adhesions in pleurisy and the "bread and butter" heart in pericarditis. He called these lesions of the vocal cords decubitus ulcers; in other words, bed sores of the vocal cords. His idea was that if you could put something in the larynx to keep the cords from coming into contact, the lesions would get well. I would like to ask Dr. Jackson whether he thinks it would be possible by the use of a modified tracheotomy tube to separate the cords and rest them in that way?

DR. NATHAN SETTEL: I have not seen any cases of contact ulcer, but wonder if it could not be a localized pachydermia over the vocal processes that has undergone secondary ulceration?

DR. C. J. IMPERATORI: From one of the granulomas an emulsion was made and this was injected into a guinea pig with the possible idea of arriving at a conclusion that it was tubercular. After the usual laboratory methods and the injection of tuberculin, the guinea pig was killed but there was no evidence whatsoever of any tuberculosis.

Insofar as the bacteriology is concerned, I feel that this conference has stimulated me to further study regarding the bacterial flora that may be the cause of this condition.

We do know that papilloma are infectious, and Dr. Landsteiner, of the Rockefeller Institute, repeated a series of experiments, re-proving what had been done in Vienna some years previously, that papilloma could be grown on mucous membranes of animals but not on the naso- or laryngeal mucosa.

DR. J. D. KERNAN: Are these ulcerations of the vocal cord? If I understood Dr. Loré, the vocal cords extend forward, and the back part of the space is the intra-arytenoid.

DR. C. J. IMPERATORI: Because of the location of these lesions, no difficulty is experienced with the voice and rarely does the patient complain of much hoarseness. Voice rest undoubtedly materially helps in the treatment.

DR. ALFRED MICHAELIS: Inasmuch as the causes given are of a general character, may not this be a trophic condition idiopathic to the individual?

DR. GEORGE D. WOLF inquired about herpes laryngis, whether it could be considered as an etiological factor in contact ulcer.

DR. J. M. LORE: Inasmuch as these lesions were all related to the vocal process, this cartilage must play an important part in the cause and course of the condition.

DR. D. H. JONES: What do you consider the first subjective symptom of laryngeal cancer?

DR. J. D. KERNAN: The first symptom is most likely to be hoarseness.

DR. D. H. JONES: How many biopsies would you do?

DR. J. D. KERNAN: I think I would take at least one other, but not more. Take a lesion which is clinically carcinoma and where the lesion involves the arytenoid fold. I said in my remarks that it should be taken at the edge with some normal tissue and reach to the base of the growth.

DR. GEORGE D. WOLF: Dr. Kernan referred to a tubercular lesion in the apex as being a cause of recurrent laryngeal paralysis. It seems to me that this is an error that crept into nearly all the standard text-books. I have discussed this question with reputable phthisiologists who tell me that this is not true. In an institution for tuberculosis averaging about 400 patients, I have examined hundreds of larynxes of patients who have all types of tuberculosis.

I can truthfully state that I have never seen a case of recurrent laryngeal paralysis. Whatever paresis may be seen in the larynx can usually be accounted for by local pathology.

DR. KERNAN: Tuberculosis of the larynx can cause a lagging of the cord—a weakening, a lack of movement in the cord. That is the way I interpret the picture where there is a weakening of the vocal cord. I spoke of a case in which the only explanation we could find was a thickening of the apex of the lung. Remember that it is not a paralysis in laryngeal cancer. It may be just a slight lagging of the cord; the cord is not as prompt to move as the one on the other side.

DR. W. J. GREENFIELD: I would like to ask Dr. Kernan's experiences with primary atrophic laryngitis. I am particularly interested in the diagnosis and treatment, as I have recently observed a case in an Italian girl, fifteen years of age. She came to the clinic because of hoarseness. Examination revealed profuse crusting over the entire larynx, including the vocal cords, similar to what one sees in so many cases of atrophic rhinitis. I thought we might be dealing with a suppurative lung condition. An x-ray of the chest, however, was negative. Likewise, x-ray of the nasal sinus was negative. There was no evidence of nasal suppurative sinus disease or of an atrophic nasal condition. She seemed to be in good general condition. Her aunt happened to be sitting near her in the clinic at the same time, being treated for what proved to be an atrophic rhinitis condition. Knowing the hereditary influence, I thought the hereditary association would make the diagnosis of a primary atrophic laryngitis the more plausible.

DR. J. M. LORE: It is important to stress the distinction just brought up, the distinction between paralysis and lack of movement; immobility is where it is due to infiltration. That perhaps would explain some extrinsic lesions of the larynx, distal carcinoma or even a distal syphilis.

DR. W. J. GREENFIELD: I would like to ask Dr. Kernan if he has seen any cases of syringo-bulbo-myelia associated with a bilateral laryngeal paralysis?

DR. KERNAN: Not that I know of; I don't think I have ever seen that diagnosis made.

DR. GREENFIELD: I have in mind a girl of eighteen years who had a syringo-bulbo-myelia with typical areas of sensory anesthesia over the shoulders, neck and chest, associated with hoarseness. She had a bilateral paralysis of the vocal cords. The true cords were both fixed in the intermediate position. I do not know how to classify this laryngeal paralysis except that it is probably a vagus paralysis not yet complete.

DR. KERNAN: That is an interesting observation and confirms what I have said concerning the necessity of a thoroughgoing neurological examination.

THE NEW YORK ACADEMY OF MEDICINE.

Meeting of March 15, 1933.

Pellagra, Oral and Pharyngeal Manifestations. Dr. Oscar Rodin.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Pemphigus Beginning in the Larynx. Dr. Herman Danish.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. IMPERATORI: The case I reported was also pemphigus of the pharynx and buccal mucous membrane. It was also seen by several dermatologists, Dr. Wise, Dr. MacKee and Dr. Cannon. One of them made some histological study. The patient never had cutaneous lesions, only those in the mouth, and esophagus. The lesions in the esophagus fused so that he practically had a total stricture of the esophagus. After two or three dilatations, this was cured, but he still has the lesions in the mouth. Naturally, one thinks that that diagnosis was not correct. I rather feel that the diagnosis I made, although seemingly proved by histological studies, was not correct and that it must have been something else, possibly a lupus erythematous bullosus.

DR. LOUIS HUBERT: A few years ago I reported a case of pemphigus of the throat. Dr. Wise also saw my case and he thought at first it was erythema multiforme bullosa, for the differential diagnosis between the two conditions is very difficult—seemingly depending upon whether the patient dies or not. Dr. Pels with Dr. Macht at the Johns Hopkins University, has developed a test in which Dr. Howard Fox has a great deal of faith. I had seen three cases that were positive by this test and all three patients died of this disease.

Cavernous Sinus Thrombosis of Otitic Origin. Dr. Jos. A. Gilbert.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Carbuncle of Nose, Ophthalmic Vein Phlebitis; Operation for Cavernous Sinus Thrombosis. Recovery. Dr. E. J. Browden.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. C. J. IMPERATORI: I think a step forward has been made in the attempt to treat cases of this type. It is likely that this man's life has been saved by this procedure.

DR. S. KNOPF: Was the external wound closed without drainage, and primary union resulted?

DR. RUBIN: Yes, it was completely closed and no drain inserted.

DR. E. M. JOSEPHSON: Will Dr. Myerson, or the reader of the paper, kindly tell how extensive was the electro-coagulation which they resorted to in this case? Also whether the objective was total occlusion of the sinus, or merely blocking its lumen.

Bullet Wound of Right Ear Canal—Hematoma of the Soft Palate and Pterygo-Maxillary Space—Tracheotomy and Ligation of Common Carotid Artery. Report of Cases. Dr. H. Rubin.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. V. C. MYERSON: There are certain details which might be added. The technique was as follows: A vertical incision was made in the temporal

region; the soft tissues were retracted; some bone was removed and the dura exposed. The dura was incised and the temporal lobe elevated intradurally. The coagulating needle was then introduced into the cavernous sinus and that structure was coagulated until there was no bleeding.

One very interesting feature of this patient's postoperative course was the fact that for the first ten days following operation he perspired profusely and constantly. It seems that the area for perspiration, which lies in the intrapeduncular region, was injured during the coagulation and as a consequence the patient perspired so much.

As has been mentioned by others, cavernous sinus thrombosis which occurs as a result of spread of infection through the ophthalmic veins usually has a concomitant orbital suppuration if the case lasts long enough. In this instance the orbital suppuration was very active. There was spontaneous rupture in at least five places, while as many other points were incised.

Another important fact in this case was its bacteriology. Staphylococcus aureus was isolated from the carbuncle and from the blood stream before operation. At the time of operation, culture, which was taken from a suppurative thrombus of the angular vein, revealed the presence of the same organism.

Peritonsillar Abscess—Retropharyngeal Abscess—Osteomyelitis of the Base of the Skull—Extra Dural Abscess and Death. Dr. Jos. S. Silverberg.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. J. M. LORE: Suppurative lesions in and about the pharynx have been of considerable interest to me for several years. Extensive cadaver and animal studies have been carried out by me in studying the spread of these infections.

One phase of this study has been the determination of the diffusion of solutions injected into the pharyngo-maxillary fossa through the anterior faucial pillar in the course of a local tonsillectomy.

If an injection is made through this pillar to a depth of half an inch, the fluid will reach the so-called pharyngo-maxillary fossa and then may travel down into the submaxillary space or reach the interior of the carotid sheath. This has been demonstrated by the use of dyes. The approach to lesions in this region is by means of the Mosher incision, which should be considered as a classic and known by all nose and throat men.

Collections of pus in the retropharyngeal space will usually extend in three directions. First, externally into the posterior triangle—back of the sternocleido mastoid muscle; second, down into the posterior mediastinum and lastly up towards the brain.

In this case under discussion, the diffusion and spread of the pus was influenced by the prolonged packing.

I feel that in this case ligation of the external carotid artery or even the common carotid artery would have been a justifiable procedure in attempting to control the hemorrhage.

DR. V. C. MYERSON: Dr. Imperatori suggests that I speak of a patient who was shown at the afternoon session today. When first seen by us he had a collection of pus which proved to extend from the nasopharynx to the lower chest. I suggested that this be opened externally, and Dr. Rubin entered the abscess from above by means of an anterior sternomastoid incision. Several tubes are in the abscess, which x-rays show to extend to the diaphragm. We are using suction every hour for one-half hour, and have converted a hopeless moribund patient into one who is definitely on the road to recovery.

DR. J. S. SILVERBERG: In reference to the bleeding: Dr. Loré asked why not ligate the external carotid? We found that the bleeding was not of an arterial nature: in fact, we were certain it was not arterial, but of a diffuse venous type. We could not exactly determine where it came from. We felt

that this bleeding was not from the internal jugular vein, but from the plexus of veins in the prevertebral region.

The speaker also mentioned deep space infection or parapharyngeal infections. Numerous cases of parapharyngeal infections with intracranial complications have been reported in the literature, but this case clinically did not look like a parapharyngeal infection and we felt that the pathway was either through the submucosa or the soft tissue.

Ludwig's Angina. Dr. Wm. J. Hochbaum.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Nutritional and Biochemical Phases of Otolaryngology. Dr. M. C. Myerson.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HENRY M. FEINBLATT: I enjoyed listening to Dr. Myerson's pertinent discussion on the important relationship between systemic indiscretions in diet and the local changes in the nasal mucosa as the result of unbalanced diets. Our greatest contributions to medicine have been made along this line of careful clinical observations and records, such procedure being of the highest scientific order.

From the standpoint of diet we recognize first the foods, such as proteins, fats, carbohydrates, salts and water; and second, accessories essential to growth and well-being classified as the vitamins, of which A, B, C and D have been best studied.

I fully subscribe to Dr. Myerson's views on the importance of maintaining a proper acid-base ratio in the diet. In speaking of acid-base equilibrium, we have in mind the availability of hydrogen or hydroxyl ions. The blood and tissues of the individual allow for a considerable increase in the ingestion of either of these without changing the hydrogen ion concentration of the blood and tissues due to the buffered state. In routine clinical examinations as reported by our speaker tonight, these variations would be too small to be appreciated from a chemical standpoint. The occurrence of such variations are not infrequent.

The tendency of the large proportion of our city-bred people is to get away from the epicurian standards of old and to get a meal that is quick and easy. The ancient epigram, "Variety is the spice of life," I mean as applies to food, is still a good one.

The internist has been keen to recognize the importance of diet and the value of dietetic control in systemic disease. Great progress has been made in the treatment of epilepsy with a high ketogenic or acid diet; the treatment of pernicious anemia with glandular tissue, such as liver, kidney and tripe; a low-caloric diet with high liver content for diabetes; liver and other glandular tissue for pellagra.

A most important group of diseases are entirely preventable by the inclusion of the proper vitamin or hormone balance. Zerophthalmia and growth deficiency by the use of the fat soluble vitamin A contained in butter, egg-yolk and in the germ of cereal grains; beriberi by vitamin B, which is water soluble and found in milk and yeast. Scurvy can be prevented by the use of vitamin C, which is also water soluble and is found in the fresh fruit juices, such as orange, lemon, tomato, fresh cows' milk and in young plants. Vitamin D is present in the fish liver, preferably in cod, and prevents rickets.

It is well to emphasize at this time that a moderate number of nutritional disturbances are difficult to trace accurately, but with a little inquiry into the habits of the individual, a definite lead can be ascertained.

The hospital I have been associated with in the past has received a considerable number of these vitamin deficiency patients from aboard ships plying between Africa, Australia and this continent. We have noticed, not infrequently, that these beriberi cases showed generalized edema and would have local nasal disturbances with neuritic symptoms.

On the subject of heredity, I fully agree with the doctor as to its importance; first, in determining the susceptibility of the individual to infection; second, in determining the locus minoris risistentia to mechanical and chemical irritation, a predisposition to allergic or atopic reactions; third, to local circulatory disturbances.

The relation of the endocrine system needs no special emphasis and I must limit my remarks here because it is so easy to go into extensive discussion. The influence of adrenalin is well known to the group here. The action of pituitrin is probably less well known.

In association with diseases of the pituitary with hypofunction, there is considerable disturbance in this local field. The low blood pressure may be a factor. This type with congested nares and obstructive breathing are often helped by small doses of obstetrical pituitrin minims three or four hypodermically. Many often show rhinorrhea. I have noticed very pronounced improvement with the use of intruitrin minims four or five. Young people who are exposed to frequent sex excitement through a long engagement often give rise to this type of nasal condition and sneezing. The use of the pituitary sex hormone has been helpful.

I feel that the paper has been inspired and will fulfill its purpose of stimulating research along these specific lines and unquestionably will lead to more rational therapeutic measures than we have at present.

DR. E. M. JOSEPHSON: I have greatly enjoyed Dr. Myerson's interesting paper. Many of our colleagues have had results similar to those described in the paper and are enthusiastic on the subject of dietetic control of upper respiratory affections.

During the past three years I have been studying a type of upper respiratory affection associated with disturbance of nutrition which possibly does not entirely fall into either the pale or red septum types. These patients complain of an incessant series of "colds," i.e., pharyngitis and rhinitis. Among other changes, these patients show transverse white streaking of the finger nails, deposits in the tympanic membrane and a relatively low blood calcium as compared with that shown after treatment. The throats of these cases generally show granular pharyngitis with massive hypertrophic, red, lymph nodules. The tonsils, when present, are generally greatly hypertrophied and show follicular and peritonsillar concretions. A number of the cases which I have studied have been associated with progressive deafness, and affections of hearing as in Dr. Myerson's cases, of a lesser gravity, were not uncommon. Among school children this disturbance has been most frequently found in the racial groups which favor very high carbohydrate diets. The symptoms respond rapidly to high doses of ammonium chloride in uncomplicated cases, that remedy which has been more or less empirically used in the treatment of respiratory affections for many an age. Ammonium chloride serves to shift the alkali reserve in the direction of acidosis. That might possibly indicate that the disorder is fundamentally in the nature of an alkalosis, which results in a fixation of calcium in the tissues. The feeding of ammonium chloride in these cases results in a rise in the blood calcium. Studies on the nature of the white streaks in the nails are under way.

DR. J. M. LORE: Has Dr. Myerson used insulin in any of his cases?

QUESTION: I would like to ask if Dr. Myerson has ever used sodium iodide in these cases.

DR. L. M. HURD: I would like to know how Dr. Myerson differentiates these red septa of diet from tobacco; and also don't forget there are a lot of red septa with tertiary syphilis. I would like to have him differentiate his pale septum from allergic septums. The pale septums, if you push them too far with the acid treatment, begin to complain of headaches and muscular stiffness.

DR. V. C. MYERSON: The interesting remarks of Dr. Josephson demonstrate that the subject is a very broad one and that we have only scratched the surface. He mentioned ammonium chloride; this is a strong acidifier.

Each grain of the salt liberates one minim of hydrochloric acid, which represents the equivalent of ten minims of the diluted acid. He also mentioned the fact that calcium was stored in the bodies of patients with an alkalosis. This is interesting and shows again how little we know, for our experience has demonstrated that calcium lactate is of value in pale septum cases.

I can answer Dr. Hurd in this way only—that each patient represents an individual study, and that we must take into consideration all the factors and indications in a given case.

Dr. Loré asked whether insulin had been tried. Insulin has been tried, but it appears that we are dealing with poor local tissue tolerance rather than poor pancreatic function in the case under discussion.

Improved Irrigating Bottle. Dr. Mervin C. Myerson.

This bottle is equipped with two valves which permit air or water to be passed through the cannula as the operator desires. There is in addition to a gauge, a pop valve which can be adjusted so that only a given maximum pressure can accumulate in the irrigating bottle. In addition there is a wire mesh surrounding the bottle so that if the construction of the bottle should be defective or if for some reason or other the bottle should not stand the strain of the pressure within, explosion of the bottle will not cause injury by flying glass.

Dr. C. J. Imperatori: It is my opinion that high pressure—over 10 to 15 lbs. air—should never be used to displace secretion from the antrum. There is always the possibility of air being distributed into the orbital or maxillary fossae. I say this for the benefit of the younger group of men who have not had much experience. Usually, every year, at the meetings of the National Societies, there are discussions of how antra should be treated without danger, and fatalities are reported. These fatalities are all traceable to the introduction of air into the antral cavities. The fatality is due to air entering a small vein, thus producing an air embolism.

Dr. L. M. Hurd: I use simply a large mouth quart bottle with three holes in rubber cork, one for the air and one for the saline, and another for the thermometer. The bottle is never opened. It is filled by suction, and pressure is made with a hand bulb.

Dr. C. J. Imperatori: Ten pounds of pressure is ample. All of these are modifications of Dr. Yankauer's idea. He felt—and he cited many cases—that there were times when no secretion came from the antrum until he put on 12 to 16 pounds of pressure. Inspissated, or very thick secretion could only be evacuated with pressure. My idea is that one must be very careful in using high pressure.